CUK-H00672-24. P16249

BRAIN:

A JOURNAL OF NEUROLOGY.

VOL. XXIV.

105
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A JOURNAL OF NEUROLOGY

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OL. XXIV.

London:
MACMILLAN AND CO., LIMITED
NEW YORK: THE MACMILLAN COMPANY
1901

Q16249

LONDON
PRINTED BY JOHN BALE, SONS & DANIELSSON, Ltd.
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BRAIN.

PART I., 1901.

Original Articles and Clinical Cases.

MENTAL WANDERING.

BY WM. JULIUS MICKLE, M.D., F.R.C.P.LOND.

Presidential Address to the Neurological Society of London, February 14, 1901.

In addressing the Neurological Society it seemed well for me to select a topic not entirely within the strict limits of insanity, to which my work has chiefly been devoted; and again, as leisure time promised to be short, one inclined to something comparatively simple and of general interest.

The subject selected is "Mental Wandering," by which I mean subdelirious conditions, delirious states, and certain dream-modifications holding definite relations thereto. The one word "delirium" may be thought sufficient; but "delirium" has been used with a considerable variety of meanings; in some countries applied to many symptom-assemblages, or even definite clinical forms of insanity; in this country usually with a much more narrow significance, but still a wide one, though shorn of elements that properly belong to it, I think. By the title, "mental wandering," fuller explanation is saved, some contentious matter avoided, and one can still freely use the word "delirium" with the meaning more usually attached to it here.

As a rule, the study of mental symptoms, especially of the less severe, must be both objective and subjective; and, like its congeners, mental wandering presents many features only known, or knowable, through the patient's self-observation. This last has obvious limitations. Nevertheless, some examples are especially suitable; the development of the subjective symptoms being in such degree and poise as to be unusually favourable to introspection, and distinctly recorded. The source of our knowledge of such cases must be the patient's account. And instead of dealing formally with the theme, it may be of more interest if we glance together at an example of this favourable kind which I have met, in the hope of finding some simple practical points and some broad truths concerning nervous and mental states, and of observing something of the passage from normal mind to abnormal.

The patient was a medical man, who, for the present purpose, will be named "A." His only mental wandering was on the occasion referred to, and a result of typhoid fever; his attacks of parotitis, measles, scarlet fever, influenza (twice), besides other febrile maladies, and brain concussion, were all free from any noteworthy mental wandering.

A number of others were infected at the same time as "A." All of them were said to have died. Thus we already meet with a practical point; the support yielded to the old view as to the existence of strong contrasts in the virulence of the infection in different outbreaks or from different sources.

The first slight deviation from health came on the night following the expiration of fourteen days from the taking of the virus, sleep on that night being defective and uneasy, and next morning a light swimming head-sensation was felt, and at first aroused idea of oncoming frontal-sinus, and nasal catarrh. For several days work was continued, and even an extra piece of night-work was done on the third night after the appearance of those symptoms. During these days and the next, malaise, weakness, and slight chilliness presaged the coming trouble; and there was something of that grey, tallowy, and stricken look sometimes seen in infections after the close of incubation and with the onset. On the fourth day the patient could not leave the house, and had raised temperature, but wrote letters, and suspected typhoid fever; and on the fifth day felt sure of it, and was so ill that he beat a retreat to bed, with high temperature, some headache, mental confusion, and budding delirium.

To-night we must not delay with the general clinical symptoms. An abstract of these forms an appendix, enabling some comparison between the general and the mental states.

But it must be mentioned now that, in comparison with the febrile movement as measured by body-temperature, the toxic element was disproportionately severe; a relation which probably had much influence on the production of the profound prostration, later profuse perspiration, and of the relatively severe nervous symptoms, manifest in insomnia, somewhat protracted though quiet, mild delirium, some peripheral neurolysis, and a variety of so-called "typhoid spine."

At starting, it is well to emphasise that much in typhoid fever is not direct pathogenic effect of its special bacillus. Sooner or later other pathogenic microbes are spurred into activity by the conditions in being, and may and do even secondarily affect the specific typhoid bowel lesions themselves. And a third important pathogenic group consists of various microbic and other poisonous substances produced in the alimentary canal in the course of typhoid fever, and all the more readily produced as part of the bowel is lamed by being the site of the specific lesions. The typhoid bacilli may be likened to swarming invaders who, besides the direct ravages they make, also loosen control in the invaded territory, and thus favour the setting free of local agents to spoil far and wide, and permit the gathering of a disaffected host in the very bowels of the land. Therefore not only many, but even the majority, of the symptoms and complicating morbid processes in an infectious disease, and especially in typhoid fever, may not be directly due to the latter's specific micro-organism, but to others, and even to some chemical products of secondary digestive or metabolic disorder. That seer in pathology, Sir John Simon, long ago limited the specificity of disease to a vegetable or animal specificity. May we not now narrow disease-specificity still more, and say that it is merely the specificity of action of the micro-organism, or toxic, or other damaging factor, which starts the first one of a given series of morbid processes—a series in which other factors or other microbes may and do play important parts?

THE MENTAL WANDERING.

On the day of retirement to bed, and for a day or two after, there were, at first some headache, but wearing off; high temperature; light-headedness; considerable mental confusion and other evidences of wandering mind—all varying in degree at different times—and recollection of parts of those days is hazy. In this light-headedness and confusion the patient was unable to keep attention on any point, and therefore did not answer accurately and relevantly beyond a few moments. The mind was thus, as dove from ark, unable to find resting place, and like to sink from memory's ken in the trackless waste of delirium.

At this juncture, owing to a misunderstanding, it fell to the patient to transmit the instructions necessary to carry out suggestions made in his presence for treatment, for disinfection, and for remodelling the bedroom arrangements and conveniences. In this state of mental wandering—by dint of effort and of frequently regaining for a moment a sufficiency of the serial order, closer reciprocal ideal relations and intimate normal ideal associations of firmer and coherent thought,—he succeeded in imparting directions; but not without *many* breaks, excursions, and repetitions. Here was a striking, but perhaps not unusual scene; a wandering-minded patient communicating the instructions for his own treatment, the mental state remaining unrecognised by those present (non-medical). Fortunately the directions so given were appropriate. From this we may draw the practical reminder to be alive to the slightest indication of mental wandering in invalids. Law may disregard the very little things; but, of them, clinical medicine must have a care.

And, of this state, another illustration occurred on the same day, when, in the course of conversation, "A" was questioned by a friend as to certain arrangements, and the latter was answered in a somewhat startling way, the bearing of his question having suffered delirious substitution, being replaced on the instant by a perverted interpretation that the question concerned arrangements for continuance of a marine structure deliriously imagined in course of erection, with mental toil and fatigue to "A," but as a project of that friend, with whom, therefore, and not with the deliriant, decision on the question ought to rest. And yet, by another turn of the wandering mind, the friend was understood as wishing the imaginary project to be carried out by "A's" instrumentality. Nevertheless, the reply made was so quick, decided, and apparently relevant; although, in truth, unfitting and surprising; that its delirious parentage was not even suspected at the time.

Yet the wavering Ego was even now partially and fluctuatingly confusing the self with others and with the surroundings. In confusion and seething turmoil, the Ego-group inclined to break up into constituent parts, to confound some of these with, or to attach them to, other persons; or to group the constituents afresh.

This is an early indication of the effects, on the systems of associational neurons, of the whole morbid operation of the disease; its toxæmia, fever, inanition, exhaustion by insomnia and over-activity. And, coincidently, degrees of motor helplessness, *e.g.*, degrees of inability for station and locomotion, marked a similar partial disablement of projection neurons, ministering to *action* in the life of external relation.

Continuing this subject of the disturbances and changes of consciousness of the self, of duple and multiple selves, and other affections of personality, it is of interest to note, thus early in the case, the peculiar confusion of self-consciousness; so that now and then the self, or some of the self's possessions, interests, rights, responsibilities, needs or attributes, became confounded in the mind of the deliriant with that or those of another, or of others; *e.g.*, the patient confused himself in some respects with his interlocutor, as on the occasion just mentioned. But when he was thus partially changed into another, or into several others, the original self could usually be re-called, more or less, co-existently.

These affections of consciousness are important. Mostly, their start seemed to be in general mental confusion. This disturbed and broke through attention; and, the serial order of ideas, and their associative inter-relations, thus broken, the mutual, reciprocal, incitant or arrestive influence of latent ideas enhanced that effect. One result was a variety of strange dissolutions of the Ego-unity-group; at first, fleeting, indistinct, partial, or, if general, incomplete; but sometimes corrected momentarily and changed by any impression sufficiently intense to rally enfeebled attention; and then, when this slackened, mind again wandering and ready to turn back into the old way, or to stray along a new one.

Thus the Ego would be partly changed into another; namely, as regards some of its relations and attributes; or as regards its mental position and *rôle* generally.

Another stage observed at times was a partial division of the self into several new ones, the basis of each and all still remaining mentally rooted in the old.

Finally, at times attaining complete partition, the self became metamorphosed into two or more several persons. Withal, the recognition of these was variable; now distinct and vivid; again receding into distance and dimness; or, anon, disappearing.

And another phase which, not long after, came, was change of the self, general but incomplete; a reduction towards the mental status of childhood, a temporary childward mental retrogression.

At one time, the deliriant seemed to be mainly substituted by three persons, of different ages, all suffering like himself, from a bad attack of typhoid fever. At this point of time "A" had refused to take beef-tea, on purely medical grounds, which at times were well within his remembrance during this time of light, quiet delirium from which he could, with ease, be aroused for the moment. And when expostulated with by the nurse, and the reason of refusal demanded; after some colloquy, his final reply was, "I'm sure it is not good for them." For *them*, be it noted. So that he was now, for the time being, assisting in the treatment of his changed and threefold self (really fourfold self); assisting, that is, by the candid expression of professional opinion; but from the view-point of the deliriant also exercising the far more effective part of forcibly modifying the diet of the imaginary three by refusing to take the actual beef-tea really offered to himself.

This may usefully remind us of the care to be exercised in noticing the least peculiarity in the expressions used by our patients. The change in the pronoun (*them* for *me*) was on that occasion the only indication of the mental wandering to the bystander. And yet it was a loophole through which apt and expert investigation would have revealed a wide expanse of delirious products largely occupying the whole range of consciousness at and about that time. The application of this is general, not individual; no medical attendant was present at the moment.

Let it be remembered that these conditions of changed consciousness of self were sometimes quickly formed and easily dissipated; occasionally underwent some transformation. They were apt to be recurrent, and in their reappearances either differing very considerably or fundamentally, or more or less similar, but never identical.

Never is the Ego twice precisely the same. In health, even, we hold and possess ourselves—each individual Ego—by no bond of absolute identity, but only by the ever-loosening ties of a relative similarity. From the adult self is ever exhaling something of the mental nature, part of the finer individuality, some of the latest gains of mental differentiation, a portion of the acquisitions and qualities of the self—difficultly renewed, or replaced by a satisfactory variant; and when decay and involution set in, renewable only in a degree, or incompletely—a renewal ever minishing of acquirements ever deteriorating.

Lest the picture seem too gloomy, I hasten to add that age has its compensations, has its usefulness. Thus the moderating regulating influence of age in council has often well served the State; so also has its strategy in the field, as witness a Moltke or a Roberts.

Affections of Nose, Mouth, Teeth, General Sensibility, in relation to Mental Wandering.

Somewhat early in the case, and continuing for a time, perverted sensibility of various parts and organs existed. Paræsthesiæ and irritation of nasal mucosa (fifth nerve) were potent sources of delirious notions. The mouth was the seat of many paræsthesiæ; the teeth felt loose and altered as if to assume a difference, not so much of locality as of semi-independence, as if they were loosened from their moorings and half free-floating in air, and yet taken to be in the mouth and mainly in their usual relations to the adjacent parts; speciously, also, communicating a feeling as if they were soft bodies, possessing sensibility. A feeling of somewhat general pulpiness of the body lasted for some days.

The perverted sensibility of the mouth and gums, the paræsthetic somewhat general pulpiness of body, and the suggestions flowing from milk diet, then in force, were eventually connected with strange delirious episodes of childish type; also connected with various false perceptions, as, *e.g.*, of coloured glass, of pressure on teeth; constituting a reversion to childhood—associations of second teething, toothache, coloured glass ornaments, cows yielding milk, and so on. Thus the reduction in this phase was to childish associations, to children's thoughts and ways of viewing these experiences.

Early marine structure-building.—Reference has been made to the structures raised on the sea or harbour of the delirium and shore adjoining. Partaking of the phantasmagorial nature of their parentage, their excitation was of a more or less shifting variable kind; and at first they were skeletal constructions of timber, a ready-made material from the impressions of youth, when the raising of such structures, and the completed open structures themselves, were often seen, and therefore were much stored away in the early strata of mind-life. And now and then, in imagination, such structures were raised, not without seeming effort on the part of the mind-wanderer.

Other phantasms.—Various illusory and hallucinatory deceptions thoroughly imposed themselves on the mind for varying spaces of time thereafter. One experience was that "A" seemed to be moving on the winding paths of unfamiliar landscape gardening. A number of hillocks or mounds were beset with pathways winding in all directions on the slopes, and ascending or descending, mounting the hillocks, or descending from their summit to the small depressions between them; and ever winding and crossing on the acclivities and declivities. And between these pathways, always winding on the steep slopes, were numerous small trees or large shrubs with unusual foliage and fruit, in fact, all of a soft pulpy appearance, reminding of waxen artifice. The colours of foliage and of the sparse fruit were subdued, many dim hues more or less brown; some gray, and some dim subdued blues and green.

At this time and thereabouts the head felt light, dizzy or vertiginoid, and insomnia was apparently complete. No sleep came to refresh. This gave anxiety to those in attendance, and often the patient, when clearer, attempted to compose himself to sleep, understanding the desirability of that restorative urged on him, and also anxious to sleep in order to avoid narcotic drugs, none of which, in fact, were taken.

This phantasmal phase seems exceedingly difficult to analyse. But the delriant's movement on winding pathways which beset the bold hillock-and-hollow configuration of the ground, this locomotion on undulating surface, and the inseparability of the two (namely, up and down movement and ground form) at the time, betoken the swaying vertiginoid state of consciousness. And certain co-incident conditions indicate a clue to further insight. For at this time there was a more or less general condition, varying in degree locally, namely, a feeling as of pulpiness of parts of the organism—an easy delirious assimilation of ideas, in the existing state of partial Ego-dissolution, endowing that false environmental appearance with something analogous to that felt attribute of the self's body pulpiness. And another co-existent condition was that of insomnia, here to be taken in relation with former experience of impairing and disordering effect of railway sleepless night travel on "A's" colour sense.

Without claiming to explain, fully, the colouration of this imagery, it certainly is interesting to note that, coincidently with the toxic and starving results of the fever and of the insomnia, modification of the colouring of the apparition was equivalent to that effected in actual sensation by reduction of the light-intensity of stimuli of colour sensation in normal condition—by failing response to the stimuli in abnormal—namely, a reduction of colour sensations more or less, on this occasion less, toward sensation of black. This would account for all but the green; which, however, may easily have been an inferential addition to the phantasm, or to the memorial image of its foliage; inferential addition analogous to that we all make, and a thousand times, in perception and memory of objects and occurrences.

We have referred to the phantasmal marine construction, also to the hillock-and-hollow episode. At a later and more advanced phase the swaying, yet mainly fluctuant and vertiginoid, state of consciousness was allegorised by the disturbed associative processes into delirious conceptions of being out on wild waters in a vessel tossed about by the waves in a regular and rhythmically recurring up and down movement, rising and falling in large undulations.

Thus it was that, when greeted on one occasion, "A" gave an account of having been out in a boat much tossed about in the seas, so that he had expected it to capsize.

This rhythmic recurrence involved (1) sensation and ideas of a particular kind of total motion in space of a comparatively passive body (although, no doubt, some fine appropriate balancing adjusting muscular tensions occurred therewith); and also (2) ideas of spatial relations;. involved, therefore, in a certain measure, what one has supposed to be a function of a certain large cerebro-cortical realm.

This regular and rhythmical affection of consciousness seems to demand a factor regularly and rhythmically recurrent at that time, and a correspondently rhythmical fluctuation in nutrition and function of the particular nervous elements concerned. And we turn to the brain circulation, naturally. So far as one could gather of the rate of apparent fall and rise, alternately, in this long billowy swell, from crest of surge to trough of sea and back again to crest, it seemed doubtful which may have been concerned of the two possible, namely, vaso-motor and respiratory, out of the three normal regular fluctuants of the brain circulation—producing waves in the blood within the great intracranial conduits. The conditions in this case were unsuitable for deciding the question, but some future example may yield the requisite relations, and that is why the question is raised here. For (if I may be pardoned a transient lapse into the hypothetical) two hypotheses at once present themselves:—(1) that toxæmia may affect vaso-motor variations of intracranial vessels so that they become morbidly mutable. Then the above rhythmic vaso-motor undulations would represent a variety of Traube-Hering waves in excess. On the other hand (2) rhythmical disorders of respiration are not difficult to produce either pathologically or experimentally, and may exaggerate respiratory undulations in brain supply.

Neither the question of the existence or non-existence of vaso-motor nerves for brain, *nor* that of abdominal vaso-motor influence on brain circulation, can be taken here.

Temporary bronchitis itself, and the effects of the inhalations used for it, were, or seemed to be, irritating; and these effects became duly translated and wrought up into various delirious notions, as, for example, of being anæsthetised or suffocated—notions which, in their turn, led to vigorous expostulation and denunciation by the patient.

The abdominal tympanites and pain of the general disease, *i.e.*, of the enteric fever, had their share in shaping the mental wandering, receiving interpretations varying from time to time, rapidly or instantaneously conjuring up conditions of suffering, of pain, of fleeting, evasive, dimly apprehended danger or attack.

Real and imagined sounds heard—such as voices or laughter—broke in strangely and unexpectedly, at times, upon the course of an ambling delirium.

But, necessarily, the *visual* impressions were a centre of much perversion. Especially when, in the flickering light of the fire in the large open fire-place opposite to the patient lying a-bed, the small china statuettes on the mantel-piece seemed to undergo a thousand transformations; retaining no set form, but as if moulded and twisted every moment by an inward plastic power, making them ever mobile, and yet always in definite relations to the transformation and movements of each other, like contortionists in lively rhythmic and incessant change, each movement of which is in unison with, and complemental to, appropriate movements in the fellow-piece of each pair. Attention of enfeebled mind, at times, was as if fascinated and held fixed by the sinuous writhings and contortions of this ceaseless, wearisome dance of two, until the eyes were kept resolutely shut or the head was turned away.

And the lights and shadows cast by the open coal fire, as it rose and fell, or threw out fiery tongues hither and thither, were in other ways a potent source of delirious conception. From time to time they brought out ideas, such as that the scene was in a dome-like edifice, in which fires mysteriously burned, and where gaily-dressed persons fluttered about; at another time, that in it was a small yacht, and in that vessel a cannon covered with flags, and the deliriant and others were taking part in a wily and successful ruse to conceal the cannon, and thus outwit the foe; or, in the dome-like space were lightly-clad figures, somewhat transformed gloriously.

Modification of Delirium by Sleep-dream, i.e., Fusion and Transmutation into Delirious Dreaming, or Dreamy Delirium.

Passing to another and later stage, we must now refer to the influence of sleep and dreaming on the clinical aspect.

After the somewhat protracted insomnia; the return of sleep, at first in a light, fitful, broken form, modified the mental state. For the wandering of mind, delirious fancies, and comparatively fleeting phantasmal appearances of the earlier period—with its insomnia, its confusion, its dizzy or vertigo-like state—became both modified and exaggerated in the dreamy states of this partial and defectively-refreshing sleep. Under *this* sleep-dream influence, the illusory and hallucinatory phenomena assumed a greater sensual vivacity; often an exaggerated, or even, sometimes, absurd expression; and there came into play more intense emotion, and wilder fluctuations in its range between the possible extremes.

I think we do not assign to dreaming states in disease all the importance they really possess, whether as symptoms, prodromic, or of the established malady; whether as modifying, enhancing, or precipitating scenes and acts in the morbid drama of delirium, or of insanity.

The phenomena of dreaming on the one hand; the illusory and hallucinatory elements of delirium on the other; have some close resemblances when viewed apart. And in the sleeping state of deliriants, such as it is, the delirium and dreams seem to fuse into one; nay, more, to be metamorphosed or even transmuted into one. Then we may speak of delirious dreaming (or of dreamy delirium).

In some dreams of disease the psychological condition is much like that of the illuded and hallucinated deliriant in waking state. Hence a resemblance to a dream-scene in the awake state.

Moreover, dreams are constituent elements in a graded scale we may construct, from the one extreme of the normal sane waking condition, to the other extreme consisting of the set hallucinations and delusions of technical insanity.

Normal sane, waking state.
Dreams in health, or fair health.
    Hypnotic and somnambulic sleep, with illusions and hallucinations.
Dreams in, so-called, ordinary disease } may unite.
Quiet delirium of ordinary disease }
Active excited delirium of ordinary disease.
Initiative delirium of technical insanity.
Episodial delirium of technical insanity.
Set hallucinations and delusions of technical insanity.

As illustrating the sometimes grotesquely exaggerating influence of delirious dreaming, it may be mentioned that on more than one occasion, when he broke out in real and profuse perspiration, the patient seemed to himself to be like to drown in, or to be carried away by the tide of, his own perspiration.

These gross exaggerations of the amount of the sweats—profuse as these were in reality—were carried over into the time of full awakening, correction being attained after the lapse of some appreciable time; another example of the points of touch between certain dreams and mental disorders, and reminder of the influence of the one upon the other.

Comparatively early, and in mid-period, had certain features presented; a recurring wearisome monotony of slow hours of delirious consciousness, a more or less painful, weary, cyclic recurrence of events, as if in a procession of the hours; each period of which had its type of morbid idea, its favourite and predominant kind, and even sometimes individualised set of ideal associations.

But chiefly was there a mental movement in an oblong quadrangular course or circuit, and not in a circle proper. With painful eagerness was it sought to carry out some act of recollection, or some imaginary work or project, or to supervise, in this circuit, whether concerning the toilsome progression of vehicle, the process of flagging the highway, the bridging and telegraph construction on the line of road; these changing from time to time, and often only vaguely remembered in particulars afterwards. The deliriant's actual conveyance, and really under his own directions, to a close-stool beside the bed seemed to him to be a triumph of engineering skill directed by him, and achieved with the aid of great personal effort and strain on his part. And here again the quadrangular element came in, apparently suggested by the square-set arms and back of the night-chair.

Later on was a frequently repeated experience. A square-set room on wheels performed a regularly recurring journey on an oblong quadrangular course. Steadily, but heavily and awkwardly, and with much shaking, concussion and vibration, did this awkward lumbering wain make its weary monotonous quadrangular round. And the wain and its processions were tacitly accepted as constituting a symbol. Though scarcely put in words, there seemed here to be a simile or an abstract summary of the experiences of that time; indeed, more than such simile, being in fact most intimately associated with and representative of the self, and a part of the existence.

The shaking of the imaginary wain was the exaggerated and altered interpretation of the real vibrations and clatter of passing railway trains, of waggons in the adjoining roadway, and of the actual demolition of a brick building; while the shape of the course of the weary processions was the same as, and apparently suggested by, the similar shape of the large bed in which the patient was lying for weeks, and of the room in which it stood, the longer and shorter measurements of the two coinciding in direction.

Thus these mental phenomena sprang from impressions issuing from the neighbourhood, fused with stereotype impress of the immediate surrounding.

Another experience concerned a structure with lines of timber, iron, and wire-rope strings, on inclined planes, rising obliquely—an instrument, vast, far-spread, and taken as analogous to a mechanism symbolic of myriads of human lives settled thereon; suggesting a prodigious, bewilderingly complex violin of human life and kind, each of the million cable-strings of which had its intense life-meaning, to touch any part of one of which indicated some individual's position and *rôle* in the social organism and in the moral and intellectual spheres, and expressed his life abstractly, in a critical summary.

Entangled on these obliquely-set cables or slanting lines of life, only able to modify the position without wholly controlling it, without the ability to alter it essentially—though by constant effort and strain some modification and improvement could seemingly be effected; and, conversely, by laxity or carelessness and effortless acquiescence, the position could worsen and tended to do so—one had here, apparently, a mental equivalent in the summarising of the deliriant's existing state of comparative physical prostration and helplessness, whilst he was lying a-bed with half-raised head and shoulders; and these conditions projecting in relief on the groundwork of a summarised sense of existence, both physical and mental.

Emotional States and Disorders in the Case.

In the production of these, also, I attribute an enhancing influence to dream consciousness (in dreamy delirium).

Comparatively calm *neutral states of emotion.* Not at all times is the mental wanderer a prey to emotion. The degrees of feeling, whether expansive or depressed, wane from time to time, and then approach or enter into what we may liken to a haven of rest shut off from the wildness of emotional storm. At these times the patient apparently becomes a more or less impassive spectator of the visions.

Expansive phases of emotion: states of exaltation.—From time to time throughout much of the wandering, the mental associations seemed, speciously, of morbid liveliness; the strict logical boundaries were easily overleaped, and much of the day-dreams, and points of satisfaction in life-history of ancestry or of the self, now and again rose to the surface.

Even amidst the weary monotony of afore-mentioned recurrent experiences were schemes and projects repeatedly conceived; and every now and then came the feeling of being able to promptly accomplish various designs; whereas, in reality, the patient was weak and helpless, incapable of getting up, or of walking or of standing for an instant. Herein is a condition to consider in relation to the morbid feeling of capability and power in mania, and often in general paralysis.

In the wide-awake consciousness of the established positive emotional phase, there was emotional and ideational expansiveness, with the degrees of quiet optimism, serene exaltation, uncurbed merriment; and with its rapid, loose, incoherent ideation. This expansiveness was influenced by the oncoming of light defective sleep, so as to be modified and transformed or transmuted into delirious dreaming; in which, while the preceding rapidity and looseness of ideation became shifting kaleidoscopic change, the emotional expansiveness of the awake state became the grandiose of the dreamy delirium, and the patient as one drunken with joy and power. In this way expansiveness attained its further degree of grandiose inflation.

Presently we come to description of this inflated period of the grandiose. But before that phase, the patient dwelt in slow transformation scenes, the scale of which was the measure of greatness. At first were glorious marble palaces of the most exquisite proportions and beauty; noble and stately temples; these triumphs of art held in the setting of lovely scenery, with hill and vale, stream and lakelet, mountain and forest, torrent and waterfall; with the far distances magnificent.

Yet, later on, there was a falling off from the serenity of this exaltation; marking, no doubt, a variation in the kind or degree of the toxæmia, or in the resistance offered to it by the nervous substance, as well as a lowering of vitality. This declension from the state of calm serene exaltation befell from time to time, and was duly weaved into the allegory of the delirium. So it was that ruin partially lighted on these stately piles with their enchanting surroundings. Tumult and quarrel and the destruction attending civil warfare, brought change of scene; some of the glorious palaces fell into ruin, others showed traces of the stress of warfare, and around them came the signs of neglect and destruction; as, for example, the wreck of what had formerly been charming wire work marking divisions of the scene, but now cast down, and lying torn and outspread in a disorder which impeded the progress, snared the feet, and entangled the movements of the passer by.

There was much difference between the scenes attending serene exaltation and certain other phenomena—speciously and superficially similar—immediately following a later part of an imaginary experience of making cold, snowy journeys in wearisome circuits (and not described in this address). For although in the latter the scenes contained grand architectural erections, these seemed to have a significance and use mainly monumental or symbolic; they were the emblems of certain influences and forces, and bound up with the personality and life-relations of certain persons more or less notable; and the difficulty in accomplishing some designs in reference to these monuments marked a crisis in existence, and symbolised failure or success. Moreover, these scenes and their columnar embellishment were far more set, fixed and stable, than those of the exaltation a few moments ago outlined. Whereas these last, indeed, were usually in some degree of change, had about them more of the spectacular than of the symbolic, marked an easier course of mental motion, a livelier fancy, which at times was dramatic in action, swift in change, fleeting in duration. Contrast with this the sober, earnest, striving effort and symbolism of the other group of scenes with imposing architectural elements.

Some time after the start of the more fully expressed expansive phase of feeling it passed into the stage of inflation already mentioned, this being somewhat variable in degree. Harmonising with the conceptions, were pervading feelings of joy, efficiency, of ease to do surprising feats, of superabundant power, actual and potential; a delirious, inflated, lightheadedness, as if the blood had been turned into glorified champagne; gay scenes of irresponsible merriment, laughter, music, merry pranks; and the swelled sense of greatness and easeful power of body and mind; in flagrant contrast with the smitten paresified reality. And then, through association of ideas, like a flash, the revelation that all this was morbid and a mockery. And then, in the burst of an imaginary chorus of laughter, the thought of "A" that he had become general paralytic, and the comment aloud recognising and naming the condition as "General Paralysis." A blundering diagnosis, but the man was then distempered in mind, and much must be forgiven him. Indeed, the mistake was no worse than some I am aware of, and without that excuse. General paralysis, with its grim, imminent fatality fully in view, and yet no shock suffered from the knowledge, no concern even felt; it scarce gave him pause from the mental rollicking and rioting in chimerical happiness. On the contrary, there was yielding to a bubbling-over merriment; this inflated viniform state reminding so strongly both of general paralytic exaltation and of alcoholic, that it may be, in part, aptly represented by a thought which was then actually in the patient's mind and phrased in the quasi-alcoholic self-exhortation, "Never mind, it's all right, *must* die *some* time."

Later came a phase in which "A" imagined himself to be whirling down the mountain slopes; or to be swiftly diving—literally diving—down through huge trees, from top to bottom, rending and peeling off thick layers of the ringed wood, and hurling them aside, as if by lightning-stroke. Yet the *absence* therewith of any delusion of being changed into a thunderbolt was grossly illogical!

These various expansive symptoms have a complex origin. They mark an unwonted and morbid incitation of, at least, large cortical areas, the substrata of ideational, kinetic and sensorial activity.

Surely with the toxæmia we have a chemical effect of transcendent fineness, working on delicate susceptibility of nervous elements, which in some examples with greater intensity of intoxication may attain to grave lesion, either by summation of effects, or through conditions promoting concentration of these locally; whereas in other examples the influence remains diffuse, light, fleeting, and the concomitant neuronic changes more or less easily recover. Sufficient to produce neuronic change is any one, alone, of several factors present: namely, toxæmia; febrile temperature; brain inanition; brain exhaustion of insomnia; to say nothing, here, of a secondary self-poisoning which may spring from each and every one of these; nor to mention the hypothetical brain anæmia.

Thus the agents are, the direct inciting, disturbing and altering effects of these several factors on function and structure of nervous elements. The accompanying disturbance of brain-circulation, impressions from the body and from beyond it, and the modification and enhancement by sleep-dream, all play their part.

On the other hand, it is in the emotionally depressed phase, yet to be described, that we find the psychical concomitants of the exhaustion and profound vital lowering of the organism and of the same poisoned neurons; a phase, as regards its extremely brief but severe manifestation, occurring later in time, although earlier, milder and transitory indications of morbid negative emotional tone had often been interposed.

The Transitory Phase of Emotional Depression.

Eventually appeared an exceedingly brief time of mental misery and despair. Nor was the advent of this entirely unprepared: the anxious straining efforts, the mental and physical malaise, and the gloomy promise existent in some of the preceding stages of the malady, had intermittently overcast the mental firmament somewhat, and prepared the way for the temporary submersion of consciousness in gloom and blackness. This, too, was the very time when the patient was emaciate, helpless, sunken in prostration, shadowed by the wings of death: nutrition at a standstill, vitality at its lowest ebb, so that for days together no gain towards recovery was made.

In this process of transient submersion of consciousness in gloomy blackness, quickly the deeps of woe were reached, than which it seemed no lower still *could* be. Then the Ego faced what was, for it, vaguely understood as a final state, a sempiternal woe and misery.

For our mental wanderer seemed to be now lying prostrated, in physical exhaustion and mental misery, amid dim and murky surroundings; and, gazing upwards, descried slowly revolving wheel-like concentric sets of metallic rings beset with numerals and other signs and figures. And it came to him as if each of these symbolised a person; and one among them was understood as being his symbol, and two others as those of two among the dearest to him in his life; while the rest were understood to represent others.

In this dim and dismal region hope was effaced, escape was impossible, and so was death; out of reach the heaven of annihilation. This misery, intense, and in prospect eternal, was taken as essential misery, and not as punishment.

Exceedingly brief, indeed transient, as was this phase of the depression, and lasting a small fraction of a day: what it lacked in duration it more than made up by intensity. It seemed as if the man was, for ever to come, fast in the pitch of the blackest pool in some grotesque mediæval inferno. And ever and above the wheeling symbols kept circling in their eternal motions.

Neither has ear heard, nor tongue uttered, the wretchedness of this state of despair.

Happily, this transient, gloomy, dismal scene may be taken as metaphorically representing the darkest hour before the dawn of a new time of continuous mental light and life.

Nevertheless, for some weeks thereafter, the continuance of helplessness, prostration, colliquative perspirations, finally with pain, made the nocturnal life, from midnight to seven in the morning, most wearisome and uneasy. But herein the predominant physical condition merely translated itself in a sombre shading of the mind—now perfectly clear intel-

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lectually, although easily fatigued, and no wonder! Indeed, like any little physical exertion, so also slight mental work, and particularly some really trivial, but now large-looming, annoyance, crossing or thwarting, increased the pulse rate, raised the temperature, resolved in perspiration; another proof, to add to the thousand already possessed, of how intimately, intricately, inextricably intermingled and interwoven are the mental and physical in man.

Contrasted with recollections of one's reading, the distinctive features of this address chiefly concern:—

(1) Partial or complete transitory division of self-consciousness, and even duple and multiple personality, in mild, quiet delirium, or mental wandering.

(2) Attempt to analyse the phantasmal experiences and apparitions.

(3) The transforming or metamorphosing or transmuting effect of sleep-dream on illusory delirious states; or of these into one.

(4) The lively, vivid, emotional play and show in mental wandering.

Minor points might be added to these, but it is better not to distract attention from the chief ones.

Subjects Omitted.

Limits of space and of time have compelled brevity and incompleteness in dealing with some, and total omission of many, of the subjects raised by this case. The omissions are chiefly as follows:—

(*a*) Further considerations on changes in consciousness of the self.

(*b*) Discussion of peripheral neurolysis in this case, also in reference to so-called polyneuritic psychosis, and to evil effect of cold, and beneficial of warmth, in toxæmia.

(*c*) The bladder symptoms, dysury, &c., and their accompanying mental symptoms.

(*d*) The constipation and dyschezia in relation to sense of effort, &c.

(*e*) So-called "typhoid spine" in the case. Reasons for rejecting the name.

(*f*) Dream-states in relation to mental disorders.

(*g*) Prominence of visual elements in the symptoms.

(*h*) The great variety of the symptoms.

(*i*) Symbolism in delirious phantasy.

(*j*) Mental wandering, as presenting comprehensive summary-sketches, foreshadowing many forms of mental disease.

In conclusion.—When we ponder on the basis of mind, it must, for us, be a lesson in humility; so imperfect the basis that mental error is more easy to understand than mental accuracy. How light our hold upon what we possess mentally; how easily that tenure relaxed or lost! A trifling disorder of health affects mind more or less, every grave disease affects it much; even in ordinary conditions the measure of mind *available at a given time* varies exceedingly; is bettered or worsened or modified by atmospheric and barometric conditions, sunshine or cloud, the state of a function or viscus, the cyclic change of day and night, quality and amount of food, kind and length of sleep, mental and moral incidents, and a thousand external circumstances. A tiny particle of many a substance can clog or disorder the working of mind, or obliterate it temporarily, and certain lesions permanently. A slight change in the chemical constituents of the blood can raise us to the height of bliss, plunge us into the depth of despair, or, as in our case to-night, with light delirious touch strike many a note in the scale of morbid mind.

APPENDIX.

At the beginning of this address the general symptoms of the enteric fever were brought down to the fifth day. A brief summary of the subsequent course of the disease is added here.

Besides the delirium, and for a time sleeplessness, the prominent symptoms, thenceforward, were: rose rash, diarrhœa, tympanites, abdominal pain, dysuria, emaciation, asthenia, profuse perspirations, some peripheral neurolysis in limbs, bronchitis, congestion of lungs, slight contracture of lower limbs, &c.

By about the thirty-fourth to forty-first day emaciation had become excessive, prostration extreme, constipation had replaced diarrhœa. Peripheral neurolysis had left some muscular wasting, motor weakness, and slight hyperæsthesia of parts of hands and feet; no improvement in general nutrition was taking place. About the forty-first to forty-third day the wise and successful restrictions in diet could be relaxed and set in train for abolition. Improvement slowly set in. About a week afterwards a variety of "typhoid spine" began to come on gradually, at first with lumbago-like pain, and, subsequently, arthralgic pain.

For a time perspiration at night had been, and continued to be, profuse, dysuria troublesome, the evenings drowsy, and the night-morning (midnight to 7 a.m.) restless and depressed; the legs still contractured, though improved by massage; and the pain-aches, and pain-shocks of lightning type, and susceptibility to chill, were still pronounced.

Throughout the two months following the forty-third day (a month-end), constipation and racking strain at stool continued; inability to walk or stand persisted, and, during the latter three-fourths of this space of time, pain on movement precluded any attempt to do so. The body continued to regain weight. The relics of peripheral neurolysis, possibly with a degree of cornual implication, consisted of atrophy and fibrillary tremors of certain muscles, some local defect of power, numbness and paresis, easily made worse by cold. At the end of that period (hundred and fourth day), the patient was taken to the seaside. A fortnight later the first step was walked. Afterwards bath-chair exercise was taken, in short turns only, inasmuch as, in the sitting position, discomfort, aching and pain about pelvis and buttocks resulted from the pressure of weight of upper part of body, even when the softest feather pillows formed the seat. Walking power gradually became re-established in the course of the following month (hundred and thirty-fourth to hundred and sixty-fifth day).

Temperature.—Unfortunately, the temperature records of the first few days are mislaid or lost; the highest point recorded during that time was 104° Fahrenheit. The charts in hand begin on the date six days after the first slight deviation from usual feeling of health. They end on the eighty-seventh day. The temperature was usually recorded six times in the twenty-four hours.

On and after the seventh to sixty-third day, the charts show a general tendency to rise in the afternoon or evening, or both; to decline considerably in the latter part of the night, *i.e.*, 2 a.m. to 6 or 7 a.m.

But there are some erratic deviations from this rule, and occasionally the temperature was moderately high (or even highest for that particular twenty-four hours) at about 11 a.m.

Seventh to twelfth days (inclusive) six days; no temperature below 100°; the range practically 100° to 102°; highest 102·5°—thirteenth to twentieth days, eight days; on a somewhat lower level again (about 1° lower), evening rises and morning falls continuing.

Twentieth to twenty-fifth days, temperature on average just below normal, and only once as high as 100°. Then on twenty-sixth or twenty-seventh days erratic rise to 101·6°, and remaining high for some hours. On twenty-eighth, twenty-ninth and thirtieth days, the temperature again down (as twentieth to twenty-fifth), then a rise to 100·6°. Then for three days the temperature constantly between 99° and 100°, day and night. Then the same (with occasional erratic rise) to the fortieth day. Then for several days subnormal mostly, and not much fluctuation.

After this several marked rises, 101°, 101·6°. Later on, subnormal for two days.

Later came tendency to late morning, midday, and early afternoon rise; but sinking in late afternoon and evening, lower and lower, and so in early succeeding morning hours, until the diurnal rise recurred (say) by 10 or 11 a.m., midday, or 1, 2, or 3 p.m., and then soon falling again.

From sixty-second to seventy-fourth days, average temperature slightly above normal, sometimes being 99° to 100° in evening.

From seventy-fourth to eighty-seventh days, temperature normal, or only very slightly above normal.

Diarrhœa: from about seventh to twenty-fourth day, and subsequently returning for a few days.

Constipation, from about thirty-third to thirty-ninth day (inclusive): enemata. Then a few days looseness of bowels, followed by two months of more or less severe constipation, requiring frequent enemata and laxatives *p.r.n.* In the month following the bowel habit was in process of becoming normal.

Urine: stated to be free from albumen and sugar at several examinations.

Diet during fever: mainly milk, eggs, and some beef-tea.

EXPERIMENTAL LESION OF THE CORPORA QUADRIGEMINA IN MONKEYS.

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In the course of our investigations upon the symptoms and degenerations following experimental lesion of the several portions of the mid and hind brain, elsewhere described (1), partial lesions of the quadrigeminal bodies and of the subjacent tegmentum were produced. It was therefore considered advisable, both from the importance of the anatomical connection of these bodies, as well as from the variable and uncertain results obtained by previous investigators, to make a further series of experiments with a view to their complete, and as far as possible limited, ablation. In the present communication the symptoms of our experimental lesions will alone be described, leaving for a future communication an account of the degenerations so induced.

The functions of the corpora quadrigemina have been the subject of many researches, but considerable uncertainty still exists as to their true physiological significance. There seems to be an inverse relation between the development and functional importance of the quadrigeminal bodies—optic lobes of fishes, amphibia and birds—and the cerebral hemispheres; and in the second place the anterior and posterior pairs have respectively different anatomical connections with the other parts of the central nervous system. Hence the experimental data may be expected to vary according as the observations are made upon lower vertebrates such as fishes, frogs and birds, or on the higher mammals such as monkeys, and as to whether the lesions affect the anterior or posterior pair or both.

From a review of the earlier literature upon the functions of the optic lobes or corpora quadrigemina, as observed by the method of destructive lesions, the results may be summarised under the following heads.

(1) *Vision.*—Various observers, among whom may be mentioned Longet (2), Flourens (3), Schiff, McKendrick (5), Ferrier (6) and Bechterew (7), found a constant relation existing between the anterior lobes and the sense of sight. Destruction of the optic lobes gave rise to visual defect, but the extent of the loss of vision was dependant upon the complete or partial decussation of the optic tracts. In those animals whose vision was monocular with complete decussation at the chiasma, removal of an optic lobe occasioned blindness in the opposite eye; while in those with binocular vision, there resulted hemianopsia to the right or left according as the left or right anterior bodies were destroyed. The experimental observations of Bechterew upon dogs are the most complete in this connection. For example, destruction of one anterior body gave rise to homonymous hemianopsia to the opposite side, and a like result followed unilateral destruction of both bodies. The pupillary reaction was only affected if the lesion extended deep into the Sylvian gray matter. A complete lesion of both anterior bodies produced complete blindness. This last observation is in harmony with one by Ferrier, who found complete blindness after destruction of both anterior quadrigeminal bodies in a monkey.

The more recent experimental observations of Fano (7A), upon testudo, and of Stefani (4) upon birds, showed that although there was some impairment of vision, as a result of destruction of the optic lobes, it was clear that vision was not entirely abolished.

The results of these experiments therefore are in favour of the view that destruction of the anterior pair, whether in the lower vertebrata, such as fishes, frogs or birds, or amongst the mammalia, such as dogs or monkeys, is followed by impairment or abolition of the sense of sight. The observations of Nothnagel (8), on the other hand, upon the effects of disease of this region in man failed to prove any interference with vision which could be ascribed to lesion of the quadrigeminal bodies as such. In a case of bullet wound of these bodies upon the right side, recorded by Eisenlohr (9), there was no affection of vision immediately after the receipt of the injury. Although the relation of the optic lobes to vision may be clear in the lower forms of vertebrate life, it is by no means so certain between the corpora quadrigemina and the sense of sight in the higher mammalia. The facts which we bring forward in this paper make it clear that ablation of the quadrigeminal bodies as such in monkeys is not followed by any impairment of vision.

(2) *Reflex action of the pupils.*—Some observers—Flourens, Longet, and others—found that the destruction of the quadrigeminal bodies caused, in addition to loss of vision, dilatation of the pupil and loss of the pupillary light reflex—an observation which indicated a centre co-ordinating retinal impressions and irido-motor action. On the other hand, Knoll (10), experimenting on rabbits, and Bechterew on dogs, did not confirm the pupillary immobility after destruction of these bodies. It was only observed by the former when the optic tract was itself injured, and by the latter if the lesion was sufficiently extensive so as to involve the gray matter around the Sylvian aqueduct.

(3) *Associated movement of the eyeballs.*—The phenomena observed upon stimulating the quadrigeminal bodies—Ferrier, Adamuk (11), Bechterew—show that, in addition to tonic spasm of the body and limbs, ocular movements of a definite character have been noticed, such as dilatation of the pupils, opening of the eyes and elevation of the eyebrows, and conjugate deviation of the eyes to one or other side. These phenomena have been ascribed by some to diffusion current, as some of the results may also be obtained by irritation of the subjacent tegmentum after the quadrigeminal bodies have been removed. On the other hand, it has been claimed that the results of stimulation are due to irritation of the quadrigeminal bodies, as such; for the

effects of irritating the anterior pair differ essentially from those consequent on irritation of the posterior pair.

This view would seem to receive confirmation from the recent stimulation experiments of Prus (18). From his experiments, which were made upon dogs without narcosis, this observer concluded that in the anterior and posterior bodies respectively are centres for influencing visual and auditory impressions, that in the anterior pair there is a centre for tonic, and in the posterior pair a centre for clonic convulsions, and that through these bodies there pass reflex paths for respiration and vasomotor action.

(4) *Maintenance of equilibrium.* — Serres in 1827, first pointed out that destruction of the corpora quadrigemina in various animals gave rise to marked instability and disorders of equilibration. These observations were confirmed by numerous investigators—Cayrade (13), Goltz (14), Kohts (15), McKendrick, and Ferrier. Thus fishes, although able to swim, could not maintain the normal attitude: frogs rolled over and over and were unable to balance themselves; pigeons, when thrown into the air, oscillated and fell down, and rabbits showed marked instability during locomotion. On the other hand Bechterew, experimenting chiefly on dogs, failed to find any disturbance of gait or locomotion after destruction of the quadrigeminal region. When these defects were present, the subjacent structures had been injured, and more especially the superior cerebellar peduncle.

A reeling gait has been described as characteristic of disease, especially tumour, of the quadrigeminal region in man. In appearance it resembles that seen in cerebellar disease rather than that of locomotor ataxy. If the disease is progressive, locomotion and the maintenance of equilibrium eventually become impossible. These symptoms have been attributed to implication of adjacent structures, rather than to interference with the functions of the quadrigeminal bodies as such. In the light of our experiments here recorded, where no ataxy or disturbance of equilibrium was observed—provided the lesion did not extend beyond the limits of the ganglionic structures—this would seem to be the true explanation.

(5) *Hearing.*—Destruction of the posterior pair was found by Bechterew, experimenting on white rats and dogs, to be followed by defect or abolition of the sense of hearing, according as the lesion was partial or complete. Only when the lesion was quite superficial did hearing seem to be unimpaired. The close association which is known to exist anatomically between the posterior pair and the trapezoid body, by means of the lateral fillet, would appear to point to an important relation between these structures and the sense of hearing. But even under the most favourable conditions it is difficult to estimate with any certainty the degree of deafness in animals; and in disease of this region in man, in which the state of hearing was specially investigated, the results are very uncertain. In the only one of our experiments (No. 2), in which the facts seemed to warrant the existence of deafness, the lesion was so extensive that not only the quadrigeminal bodies, but also the tegmentum and the central auditory tract, were destroyed. In experimental cases, therefore, in which hearing is said to have been abolished, unless a subsequent microscopic examination has been made, the direct dependance of deafness upon the quadrigeminal lesion may be open to question.

(6) *The voice and emotions.*—Some of the earlier investigators, more particularly Goltz, were of opinion that the quadrigeminal bodies are a centre for emotional expression, but the more careful observations of later experimenters have not corroborated this view. Bechterew states that ablation of the anterior quadrigeminal bodies does not produce any affection of the emotions, but that, if the posterior pair are destroyed, weakness of the voice may ensue, and that it may be completely lost if the lesion is bilateral and extensive. Ferrier, on the other hand, produced extensive lesions of these bodies in rabbits without causing cessation of the characteristic cries evoked by sensory stimulation.

Onodi (16) also has separated the quadrigeminal region from the higher part of the brain without interfering with the approximation of the vocal cords; while Sherrington (12) found vocalisation abolished after transection immediately behind the posterior bodies in monkeys.

(7) *Bodily temperature.*—Some experiments by Ott (17) upon puncture of the quadrigeminal bodies in rabbits showed a rise of from 1° to 2° F., which lasted for three or four hours. According to this observer, these effects were due to disturbance of a thermolytic centre in this locality. Irritation of the quadrigeminal region has also been found to increase the blood pressure, as well as to modify the respiratory rhythm.

METHOD OF OPERATION.

Young monkeys—*Macacus sinicus* and *rhœsus*—were exclusively employed. After deep anæsthesia was produced a considerable extent of bone was removed from the left side of the skull posteriorly and laterally, so as to expose the occipital lobe and the greater part of the angular gyrus. The dura mater being reflected, the occipital lobe was cut off at the parieto-occipital fissure, and the tentorium-cerebelli was incised back to the lateral sinus. By means of small pieces of sponge the anterior folia of the cerebellum upon both sides were carefully and gradually displaced backwards, so as to expose the quadrigeminal bodies and the veins of Galen. With care the latter could be avoided, while the ganglia were destroyed by the galvano-cautery.

In one of the earlier experiments the occipital lobe was not removed, but it was found impossible to obtain a clear view of the ganglia without removal of the occipital lobe; so that this practice was resorted to in all the later experiments, for in order to limit the lesions to these bodies, it is expedient that they should be fully exposed. Bleeding from the veins of Galen is the most serious accident which may happen. The shock of the operation is considerable and stoppage of respiration was observed on several occasions when the cautery was being applied; but further reference will be made to this.

EXPERIMENTAL OBSERVATIONS.

Experiment 1. *Macacus rhœsus.*—Immediately after the operation, the knee-jerks were obtained equal upon the two sides and of normal range. The pupils were contracted to the size of pin-points, and the eyeballs were deviated to the left side.

On recovery from the shock of the operation the following observations were made: When placed on the floor the monkey hastened off to the side of the room, the gait being perfectly steady, not the slightest tendency to fall over or stagger being detected. There was no sprawling of the limbs nor were there any tremors. On one or two occasions when startled by a loud noise he was seen to fall, but rapidly recovered his stability.

There was clear evidence of hemiopia towards the right side. As regards hearing, when a bunch of keys was jingled to right or to left, a reflex movement of the corresponding auricle was observed. There was no impairment of ordinary cutaneous sensation.

Four days later the pupils were seen to be of normal size. The eyes were moved naturally either to right or left. There was no ptosis, nor any defect in the movements of the eyelids. As far as the observation can be relied upon when tested in a monkey, the pupillary light reaction was present. The gait was steady, climbing was easily accomplished and the grasps were good. The knee-jerks were natural.

During the first few days after the operation the monkey showed a slightly paretic condition of the legs, but this did not interfere either with walking or with climbing. It passed off after the first week.

During the month the animal lived after the operation, with the exception of the right-sided hemiopia, no symptoms were detected. He was well and active, and moved freely about the cage. He was killed under chloroform, one month after the infliction of the lesion.

Autopsy.—The left occipital lobe had been removed. The cerebellum was intact. The lesion of the quadrigeminal region was practically complete. The only portions remaining were the anterior border of the nates on the left side and the posterior margin—linear in extent—of the testes (fig. 1).

Microscopically.—Examination of the sections at the level of the lesion showed that the ganglionic masses had been entirely removed, the line of demarcation passing through part of the Sylvian gray matter in the proximal parts and immediately dorsal to the posterior longitudinal bundles in the distal sections. The superior cerebellar peduncles, fillets, and internal geniculate bodies were intact.

Experiment 2. *Macacus rhœsus.*—Three-quarters of an hour after the operation the knee-jerks were obtained, but that on the left side was more active than the right. Both pupils were small, but not contracted to the size of pin-points; there was no deviation of the globes. The monkey was lively and able to move its limbs freely. He was able to raise himself without falling over or swaying. On the following day he was able to progress well, without any unsteadiness or tendency to fall. The usual tests showed that there was hemiopia towards the right side, and also a blunting of both tactile and painful sensation, and defect of localisation on the left arm and leg, and the left ear was also anæsthetic, as well as the left side of the face. There was no loss of motor power, as he was able to climb the lattice of the cage freely and to hold on tightly. There was no tremor on volitional effort.

It was difficult to be sure of the facts regarding hearing. No notice was taken of keys when jingled behind his back, nor was an auricular reflex obtained from either ear.

With the exception of a return to a medium size of the pupils—the left, however, remaining somewhat larger than the right—no change was detected in the symptoms during the ten days that the animal lived after the operation. At no period did he show any unsteadiness or ataxy.

Autopsy.—The superficial appearance of the lesion is shown in fig. 2. The left occipital lobe had been removed, and the cerebellum was intact. The lesion of the quadrigeminal bodies was the most complete of the series of experiments, no part of the superficial area of these structures being left.

Microscopically.—The sections showed that the lesion was very extensive, passing deeply into the tegmentum cruris on the right side. In this way there were destroyed the tegmentum cruris, the mesial fillet, and the central auditory tract on that side. The nucleus ruber and superior cerebellar peduncle escaped. The nucleus of the third cranial nerve was intact. The right internal geniculate body was partly destroyed, but that on the left side was untouched.

In the distal or pontine sections the extent of destruction was the same as in the previous case, the line of the lesion passing immediately dorsal to the posterior longitudinal bundles.

Experiment 3. *Macacus sinicus.*—Within a quarter of an hour of the performance of the operation the monkey was able to sit up without support. The knee-jerks were equally and readily obtained. The pupils were contracted, but there was no deviation of the eyeballs.

On the following day one was unable to observe any evidence of instability of movement, the monkey sat up without any support and showed no tendency to fall even when incited to hurry. He was absolutely steady both when progressing along the floor and when climbing the lattice of the cage, and even when sitting upon a perch.

There was hemiopia towards the right side shown by the usual tests, and for one or two days after the operation there appeared to be some defect of sensation down the right side. There was no note upon the state of hearing. The monkey remained perfectly well for one month, when he was killed under chloroform.

Autopsy.—The left occipital lobe had been removed. The extent of superficial destruction of the quadrigeminal bodies is shown in fig. 3. From this it is evident that the region had been completely destroyed with the exception of a linear zone corresponding to the hind margin of the posterior bodies.

Microscopically.—In this case the sections showed that the ganglionic masses had not been entirely destroyed in the deeper parts, and that the adjacent and subjacent structures, fillets, superior peduncles and geniculate bodies were intact.

Experiment 4. *Macacus rhœsus.*—Immediately after the operation the knee-jerks were obtained, the left being somewhat more active than the right. There was also deviation of both eyeballs to the left, and the pupils were small and equal. Owing to a considerable amount of prostration, no observations upon the gait and other functions were made until the third day, when the following were observed.

By this time the knee-jerks were equal, the pupils of medium size, and the movements of the globes unimpaired. Instead of walking across the floor in a natural way the monkey was seen to pivot upon his buttocks, but there was no instability or falling, unless he were urged. The movements of the left arm and leg appeared defective, but this was due to the anæsthesia and analgesia, which extended over the whole of the left side of the body and limbs.

The appearance of the animal suggested a careful testing of vision; and the result showed that the sight was abolished on both sides. He took no notice of pieces of apple placed before him, or of a lighted taper brought in front of his eyes. He only picked up food when it was placed in contact with the right hand. No definite facts were ascertained regarding hearing. There was no response by blinking or starting when a noise was made near him, nor any auricular reflex when keys were jingled before his ears.

At the end of one week he remained in the same condition. He groped about the floor in an aimless way, evidently because he did not see at all. There was still left-sided anæsthesia, but no instability or tremors.

During the second week it appeared as if there were some vision. He took no notice of objects anywhere to the right, but pieces of apple when held immediately to the left side of the nose were seized. There was no evidence of vision in the outer parts of the left visual fields. Towards the end of this week slight but not constant tremors were observed in the left arm on volitional effort, such as were seen in cases in which a lateral cerebellar lobe had been removed. There was no further evidence of hearing and the left-sided anæsthesia remained as before. The monkey remained without any material change in this condition for one month after the operation. By this time, however, there was evidence that he seemed to hear, for a reflex was obtained from both auricles when keys were rustled beside his ears, or when the hands were clapped in proximity to his head. The animal died six weeks after the operation.

Autopsy.—The left occipital lobe had been removed and the anterior part of the left cerebellar hemisphere was largely softened. On removing this the nature and extent of the lesion of the quadrigeminal region was apparent. This was complete, no trace of the bodies remaining. On the right side, however, the lesion extended forwards and outwards so as to invade the posterior part of the optic thalamus and the internal capsule.

Microscopically.—The sections proved the extension forwards of the lesion into the right pulvinar thalami and adjacent internal capsule. The right tegmentum cruris also was invaded, so as to involve the mesial fillet and the central auditory tract.

The explanation of the clinical phenomena in this case was therefore apparent, the blindness being due to the removal of the left occipital lobe combined with destruction of the optic radiations in the retrolenticular part of the right internal capsule and pulvinar thalami.

Experiment 5. *Macacus rhœsus.*—Immediately after the operation both pupils were found to be contracted to the size of pin-points, but after the lapse of about an hour became of normal size. There was deviation of both globes to the left side, the right eye having an inclination downwards, the left eye upwards. Occasional nystagmoid jerkings of the globes were also seen. All the ocular phenomena had passed away by the morning of the next day.

The knee-jerks were readily obtained and equal. On the next morning the monkey was again observed, when it was found that he could move his limbs freely, that there was no unsteadiness of gait nor any tendency to fall or to sprawling of the limbs. He sat quite steadily without swaying. Nothing definite, however, was made out as regards vision, hearing or common sensation.

Autopsy.—Fig. 4 shows the superficial extent of the lesion. This was incomplete inasmuch as the anterior and posterior margins of both pairs were intact. On examining the sections through the intermediate or destroyed zone, it was seen that the deeper portions had not been completely destroyed. The Sylvian aqueduct had not been opened. The fillets, central auditory tract and tegmentum were intact.

Experiment 6. *Macacus rhæsus.*—The monkey recovered rapidly, and was found to move his limbs freely; when raised he fell backwards to the left side, and was quite unable to sit up without support. The left pupil was more contracted than the right, and there was a very slight conjugate deviation of both eyes to the left. The knee-jerks were not obtained.

On the following day the animal was well and active. When placed on the floor he lay on his belly, but when urged to move on rising he invariably fell to the left side, but there was no rotation. On volitional movement, *viz.*, when food was placed between the lips, there was tremor of the head, and if the left hand was brought up to the mouth, tremors were also observed in it. There was no limb palsy, as he held on firmly both with hands and feet. There was hemianopia to the right side, but no interference with cutaneous sensation.

At the end of a week the symptoms remained the same, the most obvious being a coarse tremor of the head and body on movement, such as taking food, having the characters seen in disseminated sclerosis in man. The movements of the right arm appeared to be steady, but when the left was brought into use the characteristic "intention tremor" was noted. The gait was unsteady. The knee-jerks remained in abeyance. Ten days after the operation the monkey presented the following appearances: The left arm was maintained in a flexed and adducted position, and a rhythmic tremor observed in it, even when the animal was apparently at rest. These movements occasionally ceased, but on attempting to lay hold of food they became wild and incoordinated. The gait was unsteady, with a tendency to fall to either side. The appearances presented by this monkey were in all respects comparable to those previously seen in cases of cerebellar extirpation and lesion of the superior cerebellar peduncle.

The knee-jerks remained absent throughout. Hearing was not tested. The monkey died upon the twelfth day after the operation.

Autopsy.—In this case the left occipital lobe had not been removed. The lesion of the quadrigeminal region was incomplete. The left posterior body was completely destroyed, while that on the right side was only partially so. Both anterior bodies were intact (fig. 5). The left superior cerebellar peduncle, shortly before it entered the tegmentum pontis, was found to have been considerably injured. The tegmentum itself was not injured in any way. The microscopical sections corroborated the existence of a complete lesion of the left posterior body, while the superior cerebellar peduncle was found to be extensively injured.

Experiment 7. *Macacus rhœsus.*—In this case after the operation the pupils were found to be dilated and equal. There was a slight deviation of both eyes to the left, but no nystagmoid movements. The knee-jerks were present and equal. On the following day when more fully examined the monkey was observed to be able to walk, but tended to fall towards the right side. The gait was unsteady, and a sprawling action of the limbs was noticed. When sitting up, which he did without support, rocking movements of the body were observed. There was anæsthesia and analgesia down the right side of the body and limbs, and right-sided hemianopia. Two days after the operation well marked intention-tremors were detected in the left arm on volitional movement, such as picking up a piece of fruit, the right hand being used to steady the left. The swaying movements of the body persisted, but the general disturbances of locomotion were less. These appearances were observed until the death of the monkey on the seventh day after the operation.

Autopsy (fig. 6).—The bodies upon the left side both anterior and posterior, and the posterior body upon the right side, were found to have been destroyed, but the right anterior body was intact. The lesion, however, had extended deeply into the tegmentum on the left side. This was so extensive as almost to consist of a hemisection, the left superior cerebellar peduncle and mesial fillet being implicated.

In two other experiments (8 and 9) immediate phenomena only were observed, as the animals did not live sufficiently long to permit of further investigation. In both the pupils were contracted to the size of pin-points, but there is no note of any deviation of the globes.

In both cases during the performance of the experiment, while the bodies were being destroyed by the cautery, respiration ceased, but was restored after an interval by the performance of artificial respiration. One lived 40 hours, the other 12 hours after the operation.

In experiment 8 both anterior and the left posterior bodies were destroyed, the right posterior being intact. In experiment 9 all the bodies were destroyed with the exception of the right anterior.

Analysis of the Phenomena following Destruction of the Quadrigeminal Bodies.

The pupils.—In all the experiments with the exception of No. 7 the pupils were, immediately after the operation, contracted to the size of pin-points. In the exceptional case they were dilated, but reference to the record will show that the lesion was less one of the quadrigeminal region than of the tegmentum of the pons. This pin-point contraction was only a temporary phenomenon, for in two days at most after the operation the pupils had returned to their natural size.

As regards the pupillary light re-action, in the cases in which it was tested it appeared to be present (so far as reliance may be placed on this re-action when tested in animals, where it is so difficult to eliminate the complication of convergence and accommodation).

Ocular movements.—In all the cases with the exception of Nos. 2 and 3 the globes were deviated to the left side.

This deviation was not of more than a few hours' duration The explanation of this was not clear. The operations were all performed from the left side and with one exception the left occipital lobe was removed. Removal of this lobe, however, and the consequent hemianopsy is not sufficient to explain the deviation; nor does an unequal though bilateral destruction of the quadrigeminal region satisfactorily account for it, for it was absent in experiments 2 and 3 where the ablation was as complete as in Nos. 1 and 4, in which no deviation was noted. It was also observed in Nos. 6 and 7, where the lesion was incompletely bilateral.

It may be noted in this connection that a temporary deviation of the globes may likewise follow lesion of a cerebellar hemisphere or of the cerebellar peduncles, while we had previously observed it also in lesions limited to the tegmentum cruris.

As regards the ocular movements after the immediate phenomena had passed away, in no instance was ptosis or ophthalmoplegia detected. The absence of oculo-motor paralysis was accounted for by the fact that in no case was the lesion sufficiently deep or far forward to implicate the nucleus of the third cranial nerve.

Knee-jerks.—With the exception of No. 6 the knee-jerks were obtained throughout and equally on the two sides. In the exceptional instance they were not obtained at all, nor was any complication noted to account for their absence, with the possible exception that the monkey was in a condition of poor nutrition.

Station and locomotion.—It is in this connection that the most characteristic symptoms of gross disease of the quadrigeminal region in man are observed. We may therefore at the outset state that in the five uncomplicated cases of quadrigeminal ablation, in which the lesion was complete and bilateral, *no disturbance of stability or locomotion was observed from the time of the operation until death.* Monkeys with this lesion were able to move freely, climb the lattice work of the cage, and even to perch upon the ropes and bars.

In the two experiments (Nos. 6 and 7 in which considerable instability was detected with sprawling of the limbs during progression, falling about and tremors of the limbs and body, adjacent structures were injured. In No. 6 the superior peduncle was implicated, and in No. 7 the lesion extended deeply into the tegmentum and involved the superior cerebellar peduncle and mesial fillet.

As might be inferred from the retention of equilibrium and the absence of instability no loss of motor power was detected. In every instance the monkey grasped firmly both with hands and feet. In experiment 1 what appeared to be a paresis of the legs was observed, but it was quite transitory and did not interfere with locomotion.

Sensation.—The observations upon the cutaneous sensation were variable. But it is evident that a lesion limited to the quadrigeminal bodies, as such, was not accompanied by any impairment of cutaneous sensibility, as in the three experiments, 1, 5 and 6, where the lesion was accurately defined. In No. 2 anæsthesia, analgesia and defect in the sense of localisation was observed on the left side. In this case these phenomena were due to an extensive implication of the tegmentum cruris on the right side. In experiment 4 there was complete left-sided anæsthesia and analgesia occasioned by the extension forwards of the lesion into the right internal capsule and optic thalamus; and in experiment 7 there was anæsthesia on the right side of the body from a lesion of the left tegmentum pontis.

These observations are in agreement with what has been noted in cases of disease of this region in man, namely, that *if sensory disturbances exist they are referable to implication of adjacent structures*, such as the internal capsule or tegmentum.

Vision.—Early in the investigation it was found necessary to remove an occipital lobe in order to sufficiently expose the quadrigeminal bodies. That the right-sided hemianopsy which was present in six out of the seven experiments arose from ablation of the left occipital lobe is obvious from the fact that no additional visual defect was produced by destruction, partial or complete, of the quadrigeminal region on both sides. In experiment 4, where the monkey was rendered temporarily blind, reference to the record will show the co-existence of a left-sided hemianæsthesia, which with the left hemianopsia was due to an accidental lesion of the posterior part of the right internal capsule.

There is here therefore cogent evidence in support of the view *that destruction of these bodies has no direct influence upon vision.* If this were not so, the complete destruction which was effected in experiments 1, 2 and 3 ought to have caused total blindness.

Hearing.—Even under the most favourable conditions for testing, it is difficult to be certain as to the presence of deafness in an animal. Reliance in testing was placed upon the monkey's attention being arrested by sounds, such as the jingling of keys, at times when it was otherwise occupied, and by the presence or absence of the auricular reflex. Even in experiment 4, where the animal was blind, and the possibility of fallacy less likely, the state of hearing was uncertain.

In only one case to all the usual tests did the animal appear to be deaf, and in this instance subsequent detailed examination revealed an extensive and deep lesion of the tegmentum with implication of the central auditory tract. In experiment 1, where the lesion was accurately limited to the ganglia, there was no obvious evidence of deafness. From this it would seem that *lesion of the quadrigeminal bodies as such is not productive of impairment of hearing,* a fact which the anatomical connections of the posterior pair corroborates.

The voice.—There were no observations which led us to the belief that the voice or the emotions were affected by this lesion.

Respiration.—In two experiments, Nos. 8 and 9, in which detailed observations were not made owing to the short duration of life after the operation, respiration suddenly stopped at the time when the ganglia were being destroyed, even though the animals were well under the influence of the anæsthetic. On the employment of artificial methods the respiratory movements returned.

CONCLUSIONS.

The results of these experiments show quite clearly that destructive lesions *limited to the ganglionic masses* of the quadrigeminal region are not productive of any obvious permanent phenomena; for after the disappearance of some transitory symptoms, such as contraction of the pupils and deviation of the eyeballs, no residual effects are observed. Should such definite phenomena as instability of gait, tremors on volitional effort, anæthesiæ and defects of vision be obtained they are to be explained as the results of injury to adjacent and subjacent structures, such as the cerebellum, the superior cerebellar peduncle, the tegmentum cruris, the optic thalamus, the internal capsule, the optic radiations and the occipital lobe.

A short statement of the anatomical connections of these bodies so far as at present known, may help to explain the very subsidiary nature of their functions. Two chief sets of fibres pass to the superficial layers of the anterior pair, one from the occipital lobe reaching their destination by way of the optic radiations and the pulvinar thalami, while the other is derived from the optic tract. A few fibres from the mesial fillet appear also to enter the anterior pair. What the outgoing fibres of the anterior bodies are is not so clear. That fibres stream out and decussate in the "fountain decussation" seems certain, but the fact that there is no degeneration in the spinal cord after removal of the quadrigeminal bodies shows that descending efferent fibres do not pass directly into this structure.

The afferent fibres of the posterior pair are mainly derived from the accessory auditory ganglion of the opposite side crossing by way of the corpus trapezoides and entering the posterior pair through the lateral fillet. What has been described as the central auditory tract lies ventral to the posterior bodies as it passes upwards to the internal geniculate body, and has no direct connection with the posterior pair.

It is an interesting fact also that although the occipital corticifugal fibres terminate in the anterior bodies, no corresponding corticifugal fibres are found in the posterior bodies after ablation of the first and second temporal gyri.

It would therefore seem, when the anatomical connections are studied in association with the almost negative results of physiological extirpation, that the quadrigeminal bodies have only a very subsidiary relation to the special senses of sight and hearing.

It is difficult to understand the retention of irido-motor and general oculo-motor action after complete ablation, if the anterior bodies are the centres for co-ordinating visual impressions with the movements of the eyeballs. The only explanation would seem to be that the pupillary fibres of the optic nerve reach the third cranial nucleus by some other route than through the anterior quadrigeminal bodies, as indeed Bechterew has maintained.

We have attempted to account for the absence of deafness in strictly limited quadrigeminal lesions, by referring to the fact that the central auditory tract lies outside these ganglia, the posterior quadrigeminal body being merely an accessory structure and end station of the fibres of the lateral fillet.

The relatively small functional value of the quadrigeminal bodies in man and the monkey, as determined by these experiments, is in distinct contrast to their importance in the lower vertebrates, such as fishes, frogs and birds, assuming that the experimental lesions in these animals have been of a strictly limited character. The most likely explanation of this difference is to be sought for in the greater development and increased physiological value of the cerebral centres in the ascent of the vertebrate scale, and with this there is a corresponding diminution in their size both absolutely and relatively.

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DESCRIPTION OF THE FIGURES.

FIG. 1.

Superficial appearance of the lesion in experiment 1. It will be noted that the whole of the superficies of the quadrigeminal region is destroyed, with the exception of a small portion of the left anterior body.

FIG. 2.

The lesion in experiment 2. In this case it extended deeply into the tegmentum.

FIG. 3.

The lesion from experiment 3, showing a complete superficial destruction.

FIG. 4.

The appearance of the lesion in experiment 4. This lesion is incomplete in so far as the anterior and posterior margins of the bodies are intact.

FIG. 5.

The appearance of the lesion in experiment 6. The destruction is practically limited to the posterior pair.

FIG. 6.

Represents the lesion in experiment 7. This is also incomplete in that the right anterior body is intact, the others being destroyed.

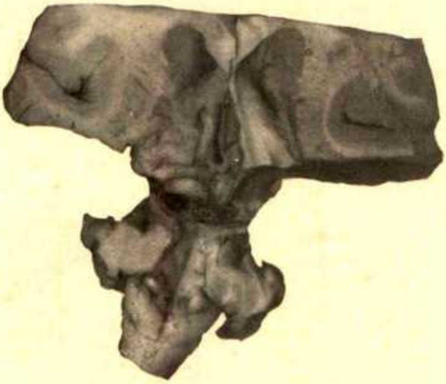

FIG. 1.

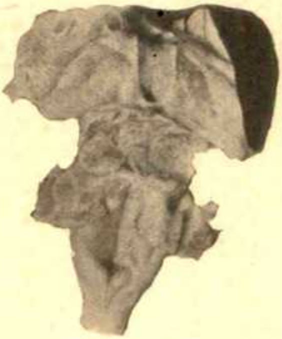

FIG. 2.

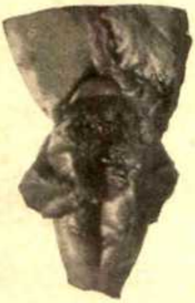

FIG. 3.

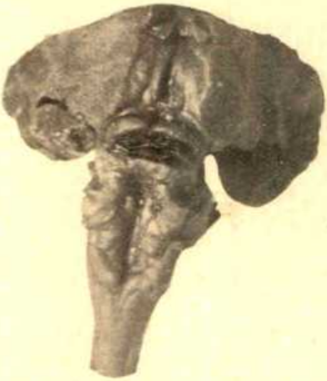

FIG. 4.

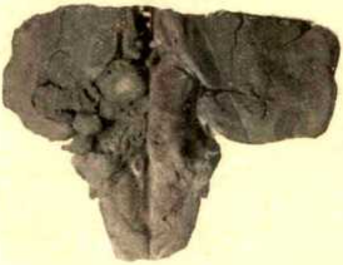

FIG. 5.

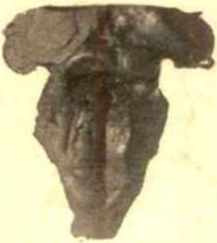

FIG. 6.

ON PARENCHYMATOUS SYSTEMIC DEGENERATIONS MAINLY IN THE CENTRAL NERVOUS SYSTEM.

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IN a "Demonstration of various types of changes in the giant-cells of the paracentral lobules," a report of which, with illustrations, is published in the *American Journal of Insanity*, vol. liv., pp. 221 to 226, October, 1897, a description is given of the findings, in a case of senile melancholia, of "changes somewhat similar to those found in the motor cells of the cord and the medulla from which the nerve process has been cut off at the periphery." A type of this lesion is seen in the cells of the facial nucleus in a case of facial paralysis due to infiltration of the periosteum of the internal auditory canal, published in the *Journal of Experimental Medicine*, November, 1897. The lesion of the cells consists in a focal reduction of the size of the stainable portions, or complete decay into dust-like particles, leaving in certain places (usually opposite the axone) an area of an almost homogeneous transparent character, which progresses to general swelling of that part of the cell body, and usually to dislocation of the nucleus to the surface. In the above case of melancholia both paracentral lobules showed lesions of the large pyramidal cells. In all of them the stainable portions were smaller than normal. In part of the protoplasm there is in many cells a homogeneous dissolution of the structure as if the details had been washed out; the nucleus frequently seemed distorted and flattened, as if pressed to the wall by this swollen portion. Independent of this, or sometimes along the margin of the swollen area, there appears an accumulation of yellowish pigment. Sections of other cells show practically no "glassy" swelling, but a peculiarity in having the very slender Nissl-bodies arranged in striking whirlpools, such as I have not seen in any normal cells, in fact, in no human nerve-cell outside of this case, and which, to my knowledge are not described anywhere. The nucleus forms in many cells the centre of these vortex formations, and frequently is so covered up by them that the Nissl stain does not give an outline of its capsule. In other cells there is a combination of glassy degeneration with whirlpool formation; in some of them apparently two distinct foci of glassy decay separated by a distinct capsule of slender Nissl-bodies. An explanation of this condition is somewhat difficult. It might be intelligible from the supposition that the glassy degeneration of part of the cell body might produce certain distortion of the internal structure in the rest, as is actually seen in many of the cells, and that cells which showed these vortex formations, would be in a state of recuperation. I would not like to be understood as considering this case as the type of what occurs in senile melancholia, since other experience in this line is not favourable to this view, and since a further examination of the basal ganglia will probably reveal lesions of the projection fibres within the internal capsules. During the correction of the proof, the following foot-note was added: "This proved to be true to a limited extent only. A second case, examined later, a patient with typical senile melancholia, gave evidence of identically the same changes as those described above. Both will be published in full as soon as the basal ganglia of the second case are examined."

During a period of three years eight cases of this type have come to autopsy. They have greatly helped to clear also the clinical side of the picture. It seems opportune to publish them at the present time, especially since publications are coming forth which in part belong to the same field, and may be furthered by a communication of our findings.

The following notes are abstracts of the histories of my cases, given rather fully, in order to allow the alienist to form an opinion of the varying mental picture, as far at least as our records furnished the data. The relatively atypical nature of most of the cases will excuse the length of some of the records, without which it would be impossible to appreciate the peculiar similarity with, and yet difference from, general paralysis. To the record of my cases I add further an unpublished case of Dr. August Hoch, and abstracts of other cases collected from the literature.

Case 1 (from Dr. Noble's service).—*Psychosis beginning like an ordinary climacteric melancholia, then passing into a condition of mutism with vague nervous disorders following a slight left hemiplegia. Three weeks before death diarrhœa; finally twitching of arms. Aged* 68 *years.*

D. S. R., born in 1829, widow, housewife. The patient's grandmother, father, two brothers and one son were insane. October, 1895, she became depressed, "she groans, wrings her hands, sways from side to side in mental anguish, worries in self-reproach for having murdered a whole family." She became sleepless, restless and noisy, refused food, and was admitted to the hospital on the eleventh day of her illness. She was fed with the tube for several days; in November she ate well; only occasionally till December, 1895, she required tube feeding; her mental condition remained about the same. She would say that she was the devil, was much depressed, rarely answered questions, and was quite inactive. March, 1896, she stayed in bed; later, during the summer of 1896, she was up during the day. She hardly ever spoke, and did not answer questions, and remained depressed and inactive, and lost flesh. October, 1896, the first entry concerning her physical condition was made. There was no evidence of dementia, but complete inactivity and mutism. The patient was always in bed. The state of nutrition was fair, but the face pale; the head large, rectangular, prominent in front; the pupils responded sluggishly to light; exaggeration of the knee-jerk is noted and ankle clonus on the left side. No reaction to prick of pin. The sleep was poor. In December, 1896, the tendon reflexes were less active; almost no knee-jerk was elicited on the right side. Bed sores began to form. January, 1897, she answered a few questions, but sometimes she did not open her eyes when spoken to, and for several days she did not swallow readily, but held the food in her mouth for a long time. This was transitory. From the answers no evidence of real deterioration was gained. There was then dribbling of urine and consequent eczema; the left arm and leg are held rather rigid, and the knee-jerk is moderate on both sides, especially on the left, on account of the rigidity. Patient takes hold of the hand, but is slow in her motions. January 25, she is said to take nourishment better and to converse somewhat more freely; "she told me this morning that she was frightened, and that she heard noises about her." End of February and in the beginning of March occasional diarrhœa was reported. March 16, 1897, twitchings of both hands and feet were noticed, and a tendency to contraction of forearms and hands, the right pupil was wider than the left; the pulse 138, weak; the respiration 40, and the temperature 100·3°. The patient was uneasy when put on her right side, and required to be moved back to the left. No definite picture of the mental condition could be made on account of the continued mutism. Death occurred March 17, 1897.

The following is the summary of the autopsy report:—

Subcutaneous ecchymoses on the forearms and over the ensiform process. Sacral decubitus. The *heart* rather large, with atheromatous patches on the mitral valve and on the beginning of the aorta. The *kidneys* slightly granular, with a number of small cysts, and sclerosed parts in the right lower pole. Weights 84 and 87 grammes. The *lungs*, with slight adhesion of the right apex, slight bronchitis in the right lower lobe, probably with beginning broncho-pneumonia. Weight of the left lung 265 grammes, of the right lung 360 grammes. Remnants of parametritis (strings of calcareous nodules). One large gall stone. *Skull*, with vortex-like *hyperostosis* on the inner plate of the frontal bone, thick. The calvaria weighs 610 grammes, instead of about 300 grammes. *Pia* œdematous, thickened and white along the longitudinal fissure. *Basal vessels* whitish. *Convolutions* somewhat atrophic.

No macroscopic lesions in the brain, except a *small scar in the right caudate nucleus*, nearly reaching the internal capsule. The spinal cord was not removed. The microscopic examination furnishes the following additional data:—

The specimen from the lung showed a number of alveoli filled with blood and with polynuclear cells, and the kidneys typical arterio-sclerotic shrinkage, with marked sclerosis of many glomeruli and their capsules. The microscopic examination of the various parts of the cortex (right and left paracentral lobules, F_2, T_1, and cornu ammonis) showed no tissue alteration, except the very marked axonal reaction of the Betz-cell on both sides, described in the first communication, and a small hæmorrhage in the cornu ammonis. The semilunar ganglion showed advanced pigmentation of almost all the cells; in many cells the nuclei are dark, in various stages of homogenisation, in others clear and large, and in a few eccentric. The stainable portions are very scanty. Sections through the basal ganglia showed that the internal capsule was not involved in the scar.

Case 2 (from Dr. Boynton's service).—*Senile depressive hypochondriacal delirium with profound emaciation and aimless restlessness. Rapid physical decay without typical nervous phenomena. Death from broncho-pneumonia. Duration of the whole process two months. Aged* 63 *years.*

J. W. H., born in 1834, shoemaker, married. His father died of apoplexy. The patient was a very conscientious worker. In 1888 he fell over unconscious (fainting?) when walking across a bridge, and was sick for two days; in 1894 he had a "fainting turn, a cold sweat came over his chest and he smelt like a dead person." Within the last two years mental efforts have become more difficult to him (such as speaking in his "lodge"); his son noticed forgetfulness for three or four years. Last winter (1896 to 1897) the patient had an attack of influenza from which he recovered imperfectly. In June, 1897, a "specialist" found his spleen enlarged. After three days of medication he became delirious; he said he was filled with wires. Reduction of the amount of medicine left him with the same somatic delusions. His wife found him a few days later out in the back yard, shoes and stockings off, carefully looking down into the toe of the stocking. "The whole of my insides have fallen out; I am trying to find them down there." In August a neighbour said something about a thing smelling bad, and the patient began to have ideas that *he* smelt bad, that he was all rotten; he said of the electric cars, "see how fast they go by now, they all smell me;" passers-by "held their handkerchiefs to their noses or went on the other side." Or he would get up at night saying, "my legs have rotted off and gone and I want to see if I can walk." He refused food because it went "to the top of his head," and on August 27 made several weak attempts at suicide by means of a twisted cloth, a shoe knife, and oxalic acid. "It is no use to live any longer." At the same time he became confused, had difficulty in recognising neighbours, was restless and did not sleep. When admitted, August 31, 1897, he only weighed 100 lbs. against 156 lbs. one year previous, and he had tremor of the hands and feet, arterio-sclerosis, prominent veins, slight enlargement of the heart, exaggerated tendon reflexes, sluggish pupils and arcus senilis. He was dazed, restlessly and helplessly standing or wandering without aims, occasionally mumbling unintelligibly; he once showed marked fear of being killed, and then could apparently not answer questions or even understand them; he did not show the tongue even when shown what was wanted. He was tube-fed from the beginning. He usually stared in a vacant manner, mouth open; once answered to the question who he was, "it was H." From September 3 he was kept in bed; restless, pulling at the bed-clothes, probably utterly disoriented. September 13: his respiration was 38, the temperature 103·8°. Hypostatic pneumonia. Death at 6 p.m.

Anatomical Summary.

Arterio-sclerotic kidneys, practically no other internal arterio-sclerosis. Heart weighing 280 grammes; flabby, but few atheromatous patches at beginning of aorta. Slight old adhesion of left lung; hypostasis of both lower lobes. Skull thin; dura adherent with small endothelioma in the temporal fossa; pia slightly thickened and œdematous, slightly whitened, with arachnoid cysts; nowhere adherent. A small lipoma in the pia of the tuber cinereum. Careful inspection of the brain revealed no macroscopic lesion in any part. No granulations of ependyma.

The cortex of the *paracentral lobules* shows the typical axonal alteration in most of the largest pyramids (Betz-cells). Among fifty there is only one normal, a few have whirlpools. The large pyramids of the third layer are not affected, except that they frequently show a decided rarefication of the stainable spindles in parts of the apex and around the perfectly normal nucleus; especially in the polymorphous layer, many nuclei are folded. In the second and third layers there are rather numerous pigmented glia cells, and a moderate number of satellites in the deeper strata. No spider-cells. The nerve-cells are moderately pigmented.

The *frontal cortex* also presents the axonal reaction in many of the largest elements. The cells are generally more pigmented and with a spongy appearance of the protoplasm; the nuclei are, however, normal. The number of satellites is a little greater than in the paracentral lobules.

The *cornu ammonis* and the *cerebellum* are normal; in the *Golgi specimens* the dendrites have a normal number of gemmules. The cells of the motor plate of the upper *cervical cord* are considerably pigmented, and many of them small and with few lumps of stainable substance. There are two instances of axonal degeneration among the twenty cells examined ; four are normal and large, two are mainly pigment bags. In several cells the lining of the nucleus is denser and its granules larger and the nuclear membrane is lost. Among the long spindle-shaped cells of the centre many have thick spindles; but one shows the axonal reaction, also one of the large cells of the dorsal horns.

The cells of the *semilunar ganglion* are of varying type; pigmentation is more moderate than in case 1, but present in most cells; the stainable substance is either present in rather large lumps uniformly throughout the cell, or much more frequently there is a marginal row of large lumps while the central part of the cell has smaller granules. In many cells the central granules are more or less hazy or even quite indistinct and the marginal lumps clotted together, and sometimes such clots of lumps form caps around the nucleus. There is probably no typical axonal reaction. The nucleus is frequently considerably eccentric; but nowhere does it break through the marginal ring of the lumps. The nucleus is normal in many cells, in others the net of granules is more dense, and all stages can be followed into the type with dark sap and absence of membrane.

In the left *lung* pneumonia with hepatisation and the diplococcus pneumoniæ is established. The *kidneys* show only moderate arterio-sclerosis. The *liver* is hyperæmic and the cells moderately vacuolised.

Case 3 (from Dr. Fleming's service).—*K. H., senile deterioration, with depression, progressing in the course of three to five years to delusions of persecution and restlessness, and finally disorientation; arterio-sclerosis. Peculiar attacks of tremor, January,* 1897; *from October,* 1897, *progressive exhaustion. Death from pneumonia, December* 18, 1897. *Age between* 62 *and* 71.

Patient is a woman 61 years old (her niece says about 70). The father died of phthisis and the mother in the latter years of her life developed a mild form of insanity. The patient was born in Ireland. She was always healthy; came to America in 1867 and worked as a domestic. Nothing is known of her sexual history; nor is there any statement concerning alcoholism. She had no school education, can neither read nor write.

Five years ago she began to show loss of memory and to worry over trifles; she was depressed, cried frequently, became retrospective; she talked and cried over things that had occurred in her early life. Two years ago she gave up work and lived alone; when her niece would visit her she would find her with but little to eat, but the patient would say that she did not want anything. Delusions developed at this time, that people were stealing and wearing her clothing. About a month before admission she boarded out, and the lady with whom she boarded was unable to keep her in the house—she would get up in the night, wander around the house, and during the day she would try to get away, saying she wanted to visit the neighbours. She became disoriented; did not realise that she was in W———, but thought she still lived in G———. (She had formerly lived there.)

She was admitted July 25, 1896, because she began to wander away from home aimlessly, and got lost. Since admission she was somewhat depressed—cried when spoken to. She removed her clothing and at times tore it, and was very restless, and her general intelligence was very defective. No delusions or hallucinations were elicited, the patient often talking to herself in an incoherent manner.

Physically she was very poorly nourished: the pupils sluggish, the knee-jerks diminished. General tremor was present at times, the grasp somewhat weakened. Dulness at the apex of right lung, without râles, but with prolonged expiration. A systolic murmur partly replaces the first sound in the mitral area. Atheroma of temporals and radials. Inguinal hernia, more marked on the right than on the left.

By August, 1896, the patient did not cry as much as formerly, answered when spoken to, but could not give a coherent account of herself. In December, 1896, she became more restless, undressing herself often, and tearing her clothing. January 19, 1897, she was unusually restless and destructive; about 11 a.m. she began to tremble all over; her mouth was drawn to the left and she was unable to speak for ten minutes; about 3 p.m. there was a similar attack, lasting only a few minutes. Nothing of the kind was observed later.

Towards May the patient became more feeble, unable to stay up long, but very restless in bed. In June, œdema of the eyelids was noticed once; but the urine was found normal on repeated examination. In July she was stronger and up; it was impossible to keep her in bed; but in October she became finally bedridden. The physical examination brought out nothing new, except that the left knee-jerk was a little more marked. In December, 1897, œdema of the feet appeared and occasional diarrhœa; the restlessness continued, the hernias became more troublesome, and, finally, December 24, 1897, the patient succumbed to a pneumonia.

The autopsy revealed general atrophy, pneumonic infiltration of the posterior part of the right lower lobe and moderate œdema of both lower lobes. The anterior surface of the right lung and both apices showed old pleuritic adhesions. The heart had very brittle and tortuous coronary arteries and there were thickened patches in the mitral and the beginning of the aorta; small, slightly cystic kidneys, partial sclerosis of the basal arteries of the brain, a few hyperostoses of the frontal bone. The brain showed nothing, except general atrophy (1,132 grammes) and œdema of the pia; no granulations of the ventricles. The other organs weighed: the spleen 40 grammes, the kidneys 70 and 75 grammes, the heart 140 grammes, the right lung 330 grammes and the left 335 grammes, and the liver 640 grammes.

Microscopically we found a pneumonia with Friedländer's bacillus, and in the cortex marked shrinkage (the œdema alteration of Hoch) and axonal alteration of the Betz-cells on both sides, also of a few of the larger cells in the first frontal gyri, but none in the temporal, third frontal, cornu ammonis or cerebellum. The sympathetic cells, however, showed much pigmentation, and almost throughout the sections a condition similar to that in case 6.

Case 4 (from Dr. Gurley's service).—*Depressive hallucinosis; after a six months' course rapid decay and fatal termination within a few weeks with features not unlike those of general paralysis, but without speech disorder or other distinct nervous symptoms. Aged* 50 *years.*

F. P. G., born in 1849, painter and agent for sewing machines. The maternal grandmother was insane after the birth of patient's mother and died insane. The patient reports of himself excessive masturbation from 10 to 12; at 15 consequent nervous debility, dizziness, inability to bear heat of the sun, two "sunstrokes" (symptoms vague); no full recovery although he was sometimes better, sometimes worse; at 20 to 25 a course of books and medicines on lost manhood, &c.; at 23 he married, and two to three months later was divorced; re-married at 25; masturbation during marriage; probably no syphilis; he was always (at least since marriage) melancholy, easily disturbed; it was hard to get him up after an illness, especially since 1894, when he had "lead poisoning" (continual constipation, pain throughout the intestines, arms, and legs, swelling of legs); he was also subject to bronchitis, and since marriage to "liver trouble" (attacks with coated tongue, retching, vomiting, and a soreness in the hepatic region). Since 1894, a change of character became noticeable, a disposition to let things drift and to spend more than he could pay for, gradually increasing to a maximum in the spring of 1898, when he bought boats, thinking he could make plenty of money. He could not pay for them and failed; he did not worry about his work; he became interested in socialistic papers and at times excited. He grew more secret and quick to take offence, would not be dictated to by his family, his wife did not dare to ask about his business. In the spring of 1898, on return from a seven weeks' trip, his eyes looked heavier; October 10 he had abdominal pains, he was fearfully depressed, thought he could not get well; that there were people around who were going to harm him; and that the doctors were going to cut him up; he thought his wife and children were against him. He was in bed until admission. From November 5 he had hallucinations of hearing: He heard his wife tell their daughter of 17 and his physician that he had laid on her too much, whereupon the doctor said that such a mean man as that should not be allowed to live; and the daughter said she did not know that he was such a mean man. Later he heard them say such a mean man ought to be killed, and they talked about having the nurse kill him. He did not believe the improbability of such conversation; he must believe it because he heard it. He remarked it was all right, and that he ought to be killed. He produced self-accusations, mainly for trivial real deeds, as thefts at 12 and 13 of money from an aunt, and his behaviour with his wife. The voices became very numerous and distressing by their threatening or disgusting contents, as "God is a whoremaster, and all the world is a whoremaster; come to meet your doom." On November 8 the patient cut his throat to forestall others harming him; November 15 he hit himself over the head with an iron; he was very changeable, at times apathetic and listless, then suddenly alarmed, or at times in deep study and averse to conversation, at other times talkative. But orientation was never affected. November 15, 1898, on admission, he was carried into the ward. "The arrest took all the strength out of my legs." He soon rallied. From the beginning he was constantly under the influence of hallucinations. Far away voices say all kinds of abominable things like "God is a fool: Frank, come here, I want you, arise, turn to the left and meet your doom," &c. He does not answer the voices but obeys them in all his actions (he took off his shirt several times, looked into the mirror to see whether he really was a beast, &c.), and frequently is at a loss: "I don't know what to do; one tells me to go up to the right, and when I did that was not right; others tell me to turn to the left, and when I did it that was not right. I don't know what to do; I wish somebody would tell me straight." Sometimes the voices are just ideas, then they become faintly audible and then louder, as if from the corridor.

The patient is a man slightly below medium height, weighing 120 pounds, with gray hair, anæmic mucous membranes and blue sclera, slight arcus senilis, but no atheroma. The wound from his attempt at cutting his throat is healing. Apart from muffled heart-sounds and a systolic murmur over the apex and left axilla and subclavicular fossa, disappearing in recumbent position, he shows much exaggeration of the knee-jerks, less of the elbow reflexes and the cutaneous reflexes, a slight fine tremor of the hands and fingers on rest and rotation, and some defect of coordination of fingers with eyes closed. Gait and speech were normal, sleep and appetite fair. The patient often sat by himself with a perplexed and depressed look; he gradually (during December) began to associate himself as travelling companion to any new and restless patient, walking up and down with him. His fear of execution varied. A voice told him to go out of doors with the others to have his bowels taken out. "I am ready to meet whatever is coming; the voices tell me my head will be cut off, the eyes taken out, the teeth pulled. I expect something dreadful is going to happen." Or he heard he was never married, the minister not ordained and the children not his, his wife dead, &c. When she called he thought she was a little stouter and her voice a little different and he doubted whether it was she. In January he appeared more indifferent to the voices and more and more devoid of affect. Orientation was always good and he showed no deterioration of memory; he had some sort of insight—"my mind is in a bad state; it keeps on thinking bad things; and I hear voices calling me to my doom." He often came to the physician with the question: "Did you call me? A voice told me to come to my doom." He took a scratch from an excited patient without resistance, "it is part of my doom." He is almost over willing to obey any instructions or orders, just as he does with the voices. Some of the voices he attributes to his own mind, and January 5, 1899, he remarked that the people around him must read his thoughts, because as soon as he thought of anything he heard it said after him. The self-accusations are meagre and are overshadowed by hallucinatory incriminations against which he remonstrates, such as having killed someone: he had only let his mother-in-law's nurse go. The idea of punishment increased. January 24 he bared his buttocks and deliberately burned them against the radiator, saying he wanted to begin on his punishment. In March, he often asked for his punishment. One day he said the food tasted metallic and he was constipated: "part of my punishment." Hæmatoma of the ears developed in April (attributed to the watchman); then he attempted to gouge his own eyes, because the voice told him to and because he was mixed up (he was ashamed of his actions); when his hand had to be restrained he tried to strike his eyes on sharp corners of chairs, faucets, &c.; he often got up at night obeying the "rise and meet your doom." Under increasing emaciation the patient finally showed a few times subfebrile temperature and vomiting, and he died unexpectedly in a collapse on May 18, 1899, after having stayed in bed four days.

The *autopsy* was made twelve hours after death and established general emaciation, residuals of hæmatoma of both ears, decensus of transverse colon and stomach, a few cysts of the kidneys, moderate adhesion of the dura to the skull, œdema and slight milkiness of the pia; the lobes were easily separated. No granulations of the ventricles. No evidence of atheroma, not even in the beginning of the aorta. General atrophy of the organs; heart 170 grammes, left lung 200 grammes, right lung 210 grammes, liver 820 grammes, spleen 50 grammes, right kidney 85 grammes, left kidney 75 grammes, brain *in toto* 1,285 grammes.

The *paracentral lobules* show no tissue alteration characteristic of general paralysis, but the axonal reaction in the greater number of the cells, in various degrees and combined with rather extensive pigmentation in many of the cells. Around the smaller cells of the deeper layers there are frequently a group of satellites. The *frontal* and *cerebellar cortex* are free.

Sympathetic (semilunar) ganglion. About one-fourth of the cells are fairly normal (large peripheral lumps and small central ones of stainable substance, and a fairly central nucleus) the majority of the cells have only a peripheral crust of stainable substance, not in distinct lumps. The centre is glassy, only occasionally laden with pigment granules, and the nucleus is more or less eccentric, in some cells reaching the surface completely as in the pictures of pronounced axonal reaction. Only few nuclei show degrees of homogenisation and slight loss of the membrane. About half the cells show pigment, but not to excess. There are no eosinophilous granules.

The *Gasserian ganglia* have large cells with fairly even and dense distribution of medium-sized and small stainable particles and rather numerous "fatigue nuclei" (condensation and beginning loss of membrane).

Case 5 (from Dr. Gurley's service).—*Alcoholico-senile deterioration. For two years periods of one to three weeks of weakness and indistinct speech. June,* 1898, *jerking of all the muscles and acute hallucinatory delirium, followed by a partial remission; from September,* 1898, *depressive delirium with somatic delusions and increasing weakness. Death from broncho-pneumonia, October* 28, 1898. *Aged* 57 *years.*

T. D., 57 years old, teamster, with good family history as far as known; with uneventful personal history, except for excessive alcoholism; he always was a heavy drinker, of whisky mostly, often taking as much as a pint daily, and he was frequently intoxicated. He married at 20, and had seven children; four died in infancy. He had not worked steadily for two years. In the summer of 1897, while working in the yard, he fell, not entirely unconscious, but with great weakness, so that he could not use either leg; understood what was said, but could not speak; he made a complete recovery in three weeks. However, from this time he had a monthly occurrence of attacks of the same kind, weakness and indistinct speech; he could not get upstairs without aid, and it was difficult to understand him. These attacks lasted one week. From the first attack the patient showed more irritability, he was hard to get along with, and drank much. June, 1898, he had jerking of all the muscles, so that he could not stand up; he acted strangely, and showed slight asymbolia, *e.g.*, he held his pipe in his hand and tried to cut it as if it were tobacco; then, about June 14, his speech became very thick and indistinct, yet he was jabbering all the time; by June 20 he had to go to bed, where he was unable to move without assistance (?). The following day there was a sudden appearance of hallucinations of sight and hearing—fairies and witches and people about the house; carpenters boring holes through the walls. He jumped out of bed, dressed, and went into the street, consulted lawyers about papers he imagined he had lost, imagined people were trying to get his property, &c. His gait was feeble and shuffling, his speech silly, careless and at times unintelligible, his attitude somewhat bewildered. He was admitted July 1, 1898. The following is an abstract of the *status* of July 9:—

Very gray thin hair, grayish moustache and beard; fair nutrition, flabby muscles, eczema on chest. Slight hypertrophy of the left ventricle. Hard radials. Marked arcus senilis, internal strabismus of right eye (from childhood), chronic conjunctivitis. Hearing very defective (watch not heard in contact); odour of oils not recognised. Tactile sense not acute. *The knee-jerks were absent*, the reflexes of the arm present. Slight tremor of hand and tongue. Bleeding hæmorrhoids. The writing tremulous and defective in spelling, not unlike that of a general paralytic, whereas the speech was merely careless. The patient was quiet and tidy in the ward, occasionally wandering restlessly, with a simple apathetic manner, and slow, somewhat unsteady gait. He was very deaf, and slow in answering, and at times rambling. Place and persons were recognised; time orientation defective—year, month, hour of the day, age, &c. Only the simplest calculations were done; but he has insight into his difficulty and in the defect of memory. Patient has a vague idea that he sometimes was pushed about—"They keep me here wrestling and I don't know what they do it for, because I am no fighting man. I have no fault to find with the folks here, only they have a good time, I suppose." His son-in-law "stole his will." He admits insight into the hallucinations at home; he hears and sees "nothing imaginary now," and "feels very silly." He ascribes his failing to drinking. No history of syphilis could be obtained.

From August 19 his appetite failed. September 15 everyone called his name at the meals. From October 4 he became restless at night, pounding the door, seeing potatoes, grass, roads, a quarry on the floor; praying. From October 11 he groaned also by day, praying, "Oh God, Oh God, Oh! I am in hell, pretty near." October 12 he jumped into a shallow pool of water on the lawn, and when his daughters visited him he prayed for their salvation, making a scene and crying bitterly. October 15, he "wanted to lick all the devils in hell, out of hell, and below hell." He became unsteady, stumbled and fell around the ward often. October 17, he was leaning over the side of his bed, coughing and retching: "The devils are into me and I am trying to get them out." After October 20, he also caused a prolapse of the rectum. "The devils have gotten down there and I try to get them out." He tore the mucous membrane, and bled. Finally, October 22, a rise of temperature and signs of broncho-pneumonia came on, and the patient died October 28.

The *autopsy* established broncho-pneumonia and œdema of both lower lobes, the left lung weighing 555 grammes, the right 830 grammes; a milk-spot on the anterior wall of the right ventricle and hypertrophy of the left ventricle (the heart weighed 365 grammes), with a few yellow patches in the mitral valve and a very few atheromatous elevations in the beginning of the aorta; a small cyst and a small scar in the right kidney (right kidney 112, left kidney 125 grammes); slight thickening of the capsule of the liver (1,470 grammes); distension of the stomach and intestine, hyperæmia of the rectum and a healing ulcer of its mucous membrane; atrophy of testicles. The skull was normal, also the dura; the pia was moderately œdematous and very slightly hazy over the blood-vessels, perfectly translucent at the base and thin over the Sylvian fossa; the lobes were easily separated; on the right anterior cerebral artery there was an atheromatous patch; the two sixth nerves were of equal size. No granulation of the ventricles. Weight of the entire brain, 1,235 grammes. The dissection of the formaline-hardened brain into the thin laminæ gave no abnormal findings, except perhaps very slight broadening of the vessels and perivascular spaces in the external capsules.

The cortex of the first frontal and paracentral lobules were examined with methylene blue after alcohol fixation. In the paracentral lobules the tissue as a whole has a normal orientation of the cells, no marked vascularity, a rather striking abundance of pigmented spindles with poorly defined nuclei in the adventitia of some medium-sized vessels, but no characteristic infiltration of the sheaths, and a moderate increase of "free nuclei," many of which surrounded with slightly pigmented protoplasm, either free in the ground substance or as satellites, and about as numerous in the layer of the medium-sized pyramids as in the polymorphous layer, certainly not more numerous around the affected cells. The first glance at the specimen shows the axonal reaction of the Betz-cell, with almost no exception and in various degrees, and modified by various degrees of pigmentation. The larger and medium-sized pyramids are not altered in the same way, but show very marked rarefication of the stainable substance of the apex process; and many of these cells and especially the polymorphous cells are pigmented and their nucleus is shrunken a little.

In the frontal cortex there are but few cells with axonal reaction; many pyramids have a very slender rarefied apex and pigmentation is prevalent. The cerebellar tissue is not altered. The *Purkinje-cells* have no axonal alteration; the spindles are plain and often form caps around the frequently irregular nucleus. In the spinal cord the *motor cells* are mostly normal, though perhaps a little pale and much pigmented; the nuclei are darkened a little in a few cells, and very few show the typical axonal alteration. The cerebellar afferent cells of *Clarke's column* are all affected and show uniformly the axonal alteration, especially well on longitudinal sections. There is a moderate number of corpora amylacea throughout the cord, but no evidence of posterior sclerosis. The cells of the *spinal ganglia* are large; not over one-third of them are pigmented. The nucleus of all the cells is without membrane, more or less dark, granular, of uneven slightly irregular outline, but with a plain nucleolus. There is a halo around the nucleus. The stainable substance of the cell body is rather heavy along the periphery, and thin or absent around the nucleus. A few cells show the axonal reaction. Among the internal organs, the *liver* shows a very slight increase of connective tissue; the *lungs* a moderately extensive infiltration with rich infection, with streptococci and staphylococci, and the *testis* has unusually open canals, but a fair amount of sperma in various stages of formation. The *kidneys* have but few obliterated glomeruli with thickened capsule; they are on the whole well preserved. In one papilla of the kidney there is a small curly fibroma well limited against the other tissue. The kidney has a few septa proceeding from the capsule and rather thick vessels, but few sclerosed glomeruli. The liver has not much blood and a moderate fatty vacuolisation of the cells in the periphery of the lobules.

Case 6.—*Recurrent (partly hysterical and hypochondriacal) depression, with several exacerbations and partial remissions; termination in stupor with some twitching, and death by pulmonary embolism. Aged* 44 *years.*

T. J. H., born 1856, painter, married. Family history: mother died of brain fever; three of the patient's cousins have been insane, also his paternal aunt, and one paternal uncle is a drunkard; a sister is melancholy. They are described as a rather nervous, excitable family. The patient at the age of 9 months injured his right hip and leg, which remained short. At the age of 14 years (1870) the left knee was crushed by a waggon. The patient went to school up to 1871, "never very bright," from childhood he was quarrelsome and cranky, and he often picked up imaginary insults. He did not get along well with his step-mother. From 1871 to 1874 he turned a wheel in a rope factory, and the next seven years he worked in a pottery. In 1878 the patient was troubled with sleeplessness and melancholia. In 1879, he was at the Carney Hospital with "symptoms of paralysis and severe 'convulsions,' and exaggerated shaking with apparent loss of consciousness and loud outcries." One night he got up from this "unconsciousness" to look in the mirror; he denied this at the time, but admitted it later. He was admitted to the *Danvers* Lunatic Hospital, September 19, 1879 ; he ate and slept well, was hysterical and lied a great deal, telling many foolish and impossible stories, and he had a series of convulsions relieved by an active cathartic. From February 20, 1880, he became more cheerful and contented, gained in weight, and was discharged *recovered* March 12, 1880.

The patient returned to his work, which he changed for house painting; this he continued up to April, 1898.

In 1889 he married, after an uneventful life of sexual abstinence, a woman of doubtful character. In 1892 or 1893 he acquired a urethritis from her; otherwise he knows of no sexual disorder, except that he failed to have erections from the Spring of 1898, while before he had cohabited about twice a week. He also mentions a lead colic in 1893, which kept him two weeks in bed. In 1896 the patient had an operation for rectal fistula. About that time he did not sleep well, and took sleeping medicine regularly since that time. He was afraid of getting sick and unable to make a living; finally, April 18, 1898, he quit work on account of depression, shaking of hands, and a pain in his bowels and lacking ambition. He became hypochondriacal, went from physician to physician, exaggerated his symptoms (*e.g.* his sleeplessness). After taking an overdose of cathartic pills in October, he began to feel that his bowels were ruptured. He was nervous, much excited, and on October 28 he had a marked convulsion of epileptic character. At home he was threatened of being strapped down for being so restless, whereupon he *cut his throat* with a razor, and was admitted here *November* 1, 1898, "for having complained of pain in his bowels, crying hysterically and for having attempted suicide."

Here the patient appeared physically normal, but for his traumatic deformities of both legs, and hypochondriacal exaggerations and prolix reiteration of his ailings, the too active motion of his bowels, and the wound of the neck, &c., the circulation in his legs—"you see, doctor, my legs are rotting off," when they were slightly blue and cold. "You will see I am going to die a terrible death."

At night the patient was frequently restless, moaning, out of bed, and requiring hypnotics.

A careful physical examination established, besides his deformities and the healing wound, exhaustion, cold extremities, a numb feeling of the legs with somewhat sluggish tactile reaction, some epigastric pulsation. The knee-jerks were equal, not exaggerated; he had a fine gelatinous tremor of the tongue, and a coarse jerky tremor of the hands at rest, increased on motion. The abdomen was scaphoid; the internal organs and the urine normal. He improved somewhat physically and also mentally, when, April 24, 1899, he was taken with a febrile tonsillitis. During the night of April 25-26 he became greatly agitated and noisy, very fearful of being killed, burned, &c. In the morning he had wild, staring eyes; he clutched the doctor, begging him not to let "them" burn him up "down there," pointing to the corners of the room; reiterating this with many groans, sobs and cries; he frequently got out of bed and kept up the same agitation all day, but with complete orientation. The temperature was 104° on the evening of the 24th, continued about 103° the 25th and sank to 100° and normal on the 26th; for some time it reached 100° on several evenings (May 25 and 26, and June 11 and 14), and occasionally in the morning (May 7 and 16); with the sinking of the temperature a quieter condition returned. But up to July 15 he maintained an undercurrent of fear with slight exacerbation of his complaints and of interpretations of the following character: a patient gave H.'s wife two letters, which she mailed for him. H. then felt "pretty sure" that he was going to be put to death. "I heard people in the dining room saying I was going to be put to death with big hatchets and an attendant said this is the last meal you will ever eat with me" (May 16). The only more complicated interpretation was maintained from May 2 until October 21, when he recognised it as imagination, but was sure he heard the talk that time: Dr. G. brought "Judge Almy" from C. to the door of the patient's room and said—"Is this the man you sentenced?" The judge replied "Yes," and Dr. G. said "that is all I want to know." This episode is referred to about April 26. H. was then sure that it was a death sentence. June 13 he said: "I am sure of it. I can see it. I can see it everywhere. I am to be taken to my room; five men will kick me with spurs and then take me to ward 'S' and string me up on the shower baths of scalding water." He at times maintained having heard, at other times having made out, that his attendants said: "Jesus Christ, when I get hold of him, I'll stitch those things into him"; or he heard that they were going to take him into Gage Hall, to take out his eyes, and "they are going to cut my testicles out the first thing." "Who said so?" "The attendants and all of them; they know all about it. They are going to chop me up to-night" (June 13). He often asked up to July—"Doctor, will you let me live another night?" or "for four days?" and then on the second day he said: "Doctor, did you mean when you said I should live over the fourth that I should not be tortured?" He realises that he was committed for an attempt at suicide, which was insane, but does not admit that his prognostications are ridiculous. He repudiates their being in any way as insane as those of B., who maintains that he is to be buried alive. His memory and orientation are good throughout, and there is at no time any foundation for his ailments. His complaints are usually produced with a mask-like countenance.

July 16, 1899, he displayed perfect insight—"They were all imaginations and I am going to brace up;" he even wanted to work; but August 20 he is again described as groaning, and weaving back and forth, slapping his hands. "My wife and furniture are all going to be lost. The probate court sits the fifth, and I am going to lose all, and everything will be swept out and my wife will be brought up here crazy." "How do you know?" "I told myself so; I made her so; no one told me." After four days of noisy lamentation, he continued depressed and restless till the middle of September. From then till November 3 he was quiet, out of doors every day, hardly volunteering anything, except occasional requests for "sleeping powder," which he did not get, or need; he, however, answered the questions promptly, was perfectly oriented as to time and place, remembered clearly all the previous conversations, called the episodes with Judge Almy "all imagination." "It was a man who looked like him, and I heard the talk about the sentence." He did not really hear the attendants say that he was to be killed; but when he heard them talking indistinctly he imagined it was about his death, "because I felt so bad." He was calm and rational, not especially depressed or anxious, only a little scared occasionally at night when he did not sleep. But he knew that it was foolish.

November 3, 1899, there was a sudden turn for the worse. An attack of diarrhœa with incontinence confined him to bed for seven days. Saliva was constantly flowing from his mouth; he often groaned and moaned: "my God, my wife is going to starve to death. She will be turned from her home." From November 10 he was up, but required bathing every day, very often asked for something (as for milk, water, to go to the closet, &c.). He spoke of the sheriff selling his household goods, but did not express any anxiety. His face became very oily and the nose especially became crusted with a simple seborrhœa, and roughened up to the forehead. November 29, the patient made an attempt at suicide. He asked to go to the closet, filled a bowl with water and attempted to drown himself in it. From November 30 he was kept in bed on account of weakness, diarrhœa, filthiness and destructiveness. He groaned and moaned often, had his mouth full of saliva and spoke almost unintelligibly, whereas before he never had shown the slightest speech-defect; he was restless, often trying to tear the bed-clothes; on several occasions he had a number of spasmodic twitchings of the arms, but they seemed to appear only when he thought himself watched. He took his food well and showed no other symptoms beyond the peculiar pseudo-stupor. January 3, 1900, his temperature was 99° at 8.30 a.m. Two hours later a rattling in the throat was noticed; the patient's temperature was then 103°; he was lying on his back with 44 respirations per minute, fixed eyes (but reacting pupils); considerable blind agitation and occasional trembling of arms and hands, especially during the forcible expiration; once or twice he drew his face in contortions, showing the teeth and lifting the arms during the inspiration in apparent distress. The heart beats were covered up by the tumultuous breathing. The radial pulse could not be obtained on account of the agitation, and the inguinal pulse was weak and irregular. The percussion of the lungs was negative. The patient flinched when pricked; both the knee-jerks were exaggerated. Death occurred at 11.30 p.m., January 3, 1900.

The autopsy, made three hours later, showed pulmonary embolism with beginning engorgement of the left lower lobe; several old adhesions of the left lung, a few yellow patches in the mitral valve and the beginning of the aorta. The weights were: left lung 280 grammes, right lung 340 grammes, heart 265 grammes, spleen 110 grammes, right kidney 155 grammes, left kidney (?), liver 1,475 grammes. The source of the embolus was searched for in vain.

The skull had almost completely obliterated sutures with the exception of the posterior end of the sagittal, a fair amount of moderately hyperæmic diploe; a moist non-adherent dura; the pia of the convexity was very slightly whitened along the blood vessels and considerably œdematous in the depending parts; the lobes separated easily; there were no granulations of the ventricles. The basal arteries were white (not sclerosed) and contained clotted blood. The weight was 1,505 grammes.

The microscopic findings were as follows:—

The sections of the *paracentral lobules* stained in methylene blue, without embedding, present a combination of acute alteration and Hoch's alteration in most cortical elements. The large pyramids have, moreover, throughout the typical axonal reaction and are little modified by the other accidental terminal alterations. The blood-vessels are not affected and the general arrangement of the tissue is normal. In the *frontal cortex* the axonal alteration is demonstrable only in the very few large pyramids. The calcarine cortex and the cerebellum are also free, and the latter not affected by the acute alteration. In the *medulla oblongata* all the cell groups showed more or less numerous instances of cells with typical axonal reaction, far in excess of what is usually found. Not only the cerebral afferent cells but even those of the nucleus ambiguus were extensively affected.

The Marchi specimens give the following findings:—The *paracentral lobules* show a large number of degenerating fibres of varying course, but mostly descending with the marrow of the convolution and appearing in or beneath the deepest layer of the cortex, and only rarely in the third layer. In the *first frontal convolution* there is only here and there an isolated degenerated fibre. The *medulla* shows scattered black lumps in the pyramidal tract, in the region of the cerebellar afferent tract, throughout the nuclei of Goll and Burdach, and among the arcuate fibres and the fillet. *But the degeneration in the pyramid is much slighter than the findings in the paracentral lobule would make one expect.*

The *cervical cord* has a marked degeneration in the posteromedian columns more on one side than on the other, few lumps in the postero-lateral column, more in the lateral columns and in the direct pyramidal tract and relatively few in the anterior ground bundle.

Case 7 (from Dr. Fleming's service).—*M. D., a woman of* 33 *years, of French-Canadian origin. Melancholia with marked perplexity, leading to a peculiar delirious agitation with twitchings* *and attacks of fright during an irregular partly febrile episode of enteritis lasting nearly four weeks.*

The maternal grandmother of the patient is 106 years old, and has been peculiar mentally in her old age. *The oldest child* of the patient is an epileptic imbecile. The other children are normal, also four brothers of the patient. The mother died in December, 1899, of unknown cause. The patient was born in Canada, 1867. She went to school very little, if at all, and was obliged to weave in a mill at the age of 11. She was rather weak physically (on account of the early work) but normal otherwise. At the age of 21 she married Mr. D., after two years' acquaintance; of very retiring disposition, she devoted herself to the housework and the children, and to learn to read was her only pastime. Her husband calls her an intelligent and good housekeeper, of temperate habits and cheerful, never gay and never despondent.

Two of the patient's brothers of 12 and 22 years boarded with her; two other brothers lived in the neighbourhood. In 1896 her husband became a Methodist and they began to read the gospel, and the wife and two younger brothers were also converted. The two other brothers would often come to the house and worry their sister by telling her that their grandmother, "who was a witch," had given her the imbecile boy and had changed her religion.

On December 21, 1899, the patient's mother died. From this time the patient became greatly depressed, cried often, and finally almost incessantly day and night for four or five weeks. She would say "my mother is dead and my grandmother too" (she had only heard that she was weak), and she would cry out "I am damned," because the grandmother was dead. Up to February 2 she did all her work, except three weeks' washing which she neglected. She thought she saw her mother in dreams, but she did not hear her speak, nor did she have any hallucinations when awake. During this period her brothers persisted in coming to the house during the husband's absence, and in worrying the patient about her conversion.

Anxiety began to make the patient sleepless. She would not go to bed. She heard people come in downstairs, and although she did not hear them speak she knew they wanted to kill her. "As soon as I fall asleep they will come up." Hence she sat up most of the night. During the night of February 23, 1900, her husband saw her jump from the second storey window, but succeeded in catching her arm and in pulling her in. She was very much excited, she "had married the devil, and she and her children were to be killed." In a paroxysm of anxiety she had tried to commit suicide, and it was impossible to quiet her. She was taken to the Poor Farm the next day. She was locked up and restrained several days, wanted to go home, but became more quiet, and passed into an attitude of timid apprehensive bewilderment. She never spoke to any of the inmates, but often picked their clothing and followed them around. She would stand by the matron from five to thirty minutes without saying a word. When her relatives came she recognised them, but would only sit by them holding their hands, looking at them in a puzzled way, and would hardly say more than four or five sentences in an hour; she wanted to go home, objected to being shut up, and said that she would do better at home. She did not know clearly where she was; she was not untidy, but required dressing and undressing.

On April 4, 1900, the patient was admitted to the hospital. She was a woman of about five feet, weighing only about 87½ pounds, hair brown, rather thin and fine, eyes gray, complexion sallow. There were several ecchymoses on the left leg and arm and around the left eye. The palate shallow. The lobules small and adherent; the right lobe of the thyroid somewhat enlarged. The recti abdominis muscles were separated, and there were numerous lineæ albicanthes. No disorders of the senses were discovered; the knee-jerks were brisk, the wrist and elbow reflexes present, the plantar reflex normal; ankle clonus and abdominal reflexes were absent. There was a slight tremor of hands on rotation and a slight gelatinous tremor of the tongue. No other motor disorders. The internal organs showed nothing abnormal. The urine was reddish-yellow, somewhat clouded, acid, of specific gravity 1030, free from albumen or sugar; no casts.

On April 5 the patient was found sitting on a settee. When spoken to she looked with an expectant half-puzzled expression without fear. To repeated questions she said she was fairly well, and spelled her name; she was difficult to understand, as she spoke hesitantly and hardly above a whisper, and in very poor French. She was led into a room and she sat down with a simple inquiring expression, slightly parted lips and sleepy eyes. While this description was dictated she remarked: "They brought me in a carriage." When smiled at she smiled. "Where do you live?" "I live here." "What is this?" "I don't know; I don't know all the rooms. Doctor, have I got to go into all the rooms here?" "What kind of a house is this?" "It is here." "What do you call it?" After it was explained she was asked what had been said. "You said the hospital." "Where?" "In Worcester." "Are you ill?" She hesitates, looks around uncertainly, and finally says after several starts: "I am not ill." "Is your head troubled?" After long hesitation and numerous repetitions of the question, she whispered "no" to the side with a slightly embarrassed negative expression. "What did I say this was?" After many repetitions of the question, she said, "The hospital of St. John, you said." When the physician left her, she also left her chair in a simple, contented, though uncertain, hesitant manner. The general picture was that of a poorly-nourished woman, who is puzzled, uncertain, not able to grasp the situation for any length of time, but evidently anxious to do something. She follows the attendants with a perplexed look. The emotional tone is rather indifferent.

April 12, she presented very much the same picture: she is hesitant, perplexed, looks around puzzled, but remembers the writer. "Yes, I think I have seen you before." "Where?" "I was in a room with the mistress of the house (pointing to Dr. F.). Is she the mistress of the house?" "What house is this?" "I don't know, they took me here." "What did I tell you it was?" "You told me" (here she paused and began to cry a little) "You told ~~me~~—they took my ring." "Who?" "A woman that brought me here. They brought me here to make me live. I would like to have my shawl," pointing to her shoulders. "In what city are we?" She looks around studying, breathing deeply, and after many questions she mumbled something unintelligible. "Are you in Canada?" She brightens up a little and finally says, "Yes." "Can we do anything for you?" Here she begins to whimper and finally mumbles something. "Do you need anything?" "Make me live." "Are you ill?" No answer. "Are you dead?" She smiles and says: "I don't want to die either." "What do you want?" "I want to stay with those people (*créatures*) out there, with the people" (*monde*). "Are you afraid?" "If one does not do anything, I'm not afraid, but if one does something, I am afraid." "Why are you so sad?" She begins to breathe more rapidly, whimpers a little and says, "I want to—I want to—" Then she pulls out her handkerchief and blows her nose on the extreme corner. "Do you need anything?" "No." The *perplexity* and *hesitation* and *slight uneasiness* are plainly in evidence. The answers were difficult to elicit, but when the patient once spoke, she did so without retardation.

April 19 and 26, the picture was similar. In the meantime she ate fairly well, but lost flesh; she weighed but 78½ pounds. On April 30, she had to be helped in undressing and resisted somewhat; usually she sat quietly on a settee with an expectant half anxious face, and occasionally worry, or she followed the attendants, or was anxious to have them sit down beside her. At times she suddenly jumped up, shook her dress and assumed a frighted expression. On the 19th she was greatly perplexed and uneasy; sitting on the chair with the physicians, she leaned over to the left side while slowly peering around, then made symmetrical movements with both hands around her waist, and then down the thighs and legs. "What do you want to say?" "I am all right" (without changing her puzzled attitude). "Please go and shut the door." She turns around slowly, still leaning to the left, turning her head to the right to the door. When requested again, she gets up slowly and awkwardly, and remains standing near the door; after about a minute she puts her hand on the door knob and draws the door open; then turns puzzled to the physician, wipes her face and takes hold of the door again. "Come and sit down!" She opens the door again. "Now close the door and come and sit down!" She leaves the door open and comes sideways with slow steps and sits down. "You know this house?" "Yes," but nothing could be elicited. "Have you pains?" "Why do you look so?" Her eyes became a little flushed and the face more troubled. "Can I do anything for you?" She begins to breathe more hastily and looks as if she would begin to cry. "Well, what have you to say?" She looks puzzled, the head slightly stretched forward, makes a few starts as if to speak, but remains silent. On April 26, she had the same expression of extreme uncertainty, reached out her hands in an uncertain half ataxic way and held the hand; she said: "This is the hospital." "And what is it called?" "My name is Mathilde D." Beyond this nothing could be obtained.

On May 10 she appeared equally hesitant and perplexed. "Do you feel sick?" "I am—I am—I am—I am not sick; I don't want any medicine. They make me take medicine and it does not do me any good" (making motions with the hand over the stomach).

On May 17, she had been in bed three days with a cold. "How are you?" "I have a cold" (rhume). After this frank response she shut her eyes, would not answer, and resisted slightly.

On May 11 her temperature was 99·8°, from May 12-13 101° to 101·6°, from May 14-22 99° in the morning and 100° in the evening. The appetite was poor; food was taken on persuasion only, and the sleep began to sink from eight hours to almost nothing on the 23rd and 24th, when the temperature rose above 102° and in the evening to 104·6°, to sink to the normal again May 25-29. May 20: a blood count was made showing 4,000,000 red and 6,500 white corpuscles. Diarrhœa came on; in one of the five stools of May 22 there was some blood. The abdomen became slightly distended and occasionally a little painful to touch. May 23: the blood count was the same (4,000,000 and 6,500), with a temperature of 103. The diarrhœa was arrested until May 28, and later reappeared irregularly. Widal's test was negative (May 25).

The *mental condition* became that of a *perplexed agitation* with occasional attacks of fear and screaming. May 23, when her *husband visited her*, she first cried "eh—eh—eh," &c., stretched arm and hand against him and turned the head away, but then at once took his hand and kissed him. She knew him but asked no questions and answered none except "Are you in L. or in W.?" "In this little corner." "Do you worry about the children?" "No." She often said "T." (the name of the brother who had troubled her), and at times cried and screamed, and tossed the arms around. She called the husband back but had nothing to say. May 24: she was found with dry, cracked lips, sordes, but moist tongue, with a number of aphthæ, somewhat distended, tympanitic and gurgling *abdomen*, hardly sensitive to palpation; the skin of the chest and arms red and scratched from the restless friction and picking. She makes uncertain, unsteady movements with the hands, mostly symmetrical, often with one finger down on the body or lips, moving along tremolo-fashion or making a poorly-defined picking movement. The facial expression varies; a smile she answers with a sardonic smile. She pays prompt attention but *from time to time she gets the expression of fright*, with increased restless, partly defensive, movements of the arms, and a stare and scream. "Yes." "What do you want?" "I want Caspar." "Do you want Caspar?" "Yes." "Your brother?" "Yes." "Do you need anything?" "Oui,"—long drawn out, while she puts her fingers to the lips, and ending in a scream and a movement of the arms to cover the face. "What do you need?" "I need—I need nothing," again in a calmer, half-whispering tone. When asked to show the tongue she said "oui," answered a smile, then began to stare, and screamed and struck at the physician, looked away crying "docteur, docteur," and was quiet again. After the tongue and throat were examined, under slight resistance, she was asked, "Are you afraid?" "Yes, oui, oui," and again she screamed, staring with a slight *nystagmus* and ending up with a "non-non-non-non-non," returning to her palpation movements. "Do you know me?" "Yes." "Who am I?" She looks and finally says "no," and "I am afraid of you," with a new attack of fear which was rapidly pacified. Later she again answered a smile. She often repeats a few words of a question or answers "oui" without always grasping the sense. To a repeated "Who came to see you yesterday?" she said "Nobody." "Your husband?" "Yes." "Are you sad?" "Yes." "Why?" "I don't know," just with the lips. Then after a long pause, with movements of the hands and lips and another screaming attack, she turned to Dr. F., saying, "bonjour, bonjour." No orientation; she does not shake hands when the physician leaves her.

From May 26, she was more quiet, slept more, and took food and medicines better; she still had from one to five movements most days. The pulse remained about 110. On May 31, although the temperature had again risen above 100° and 101°, she spoke rather more freely and was less frightened. "Good morning; how are you?" "Good morning; I am glad to see you." "Do you want to take me away?" "I have 2,000 piastres at home; my father is dead, but he left me 2,000 piastres. Won't you take me up to X. (unintelligible)? I want to get out of here." "What is this?" "A hospital." "Where?" She looks to the ceiling and answers: "I am going to give you money if you take me away." To many questions whether she is sick, she answers nothing. She shows the deep red, slightly parched tongue, but gets it hardly beyond the teeth. "Do you need anything?" "Yes, I want to eat." "What city are we in?" (repeated)—no response, nor to the question whether the husband had been here, who the physicians were, whether palpation of the abdomen hurt. She mumbled several times something like "I want to go away." "Have you pain?" "No." No speech disorder was established at any time.

The first week of June she grew more sleepless; she ate poorly, cried often, and from June 6th she began to show a peculiar twitching of arms and body, with occasional twists of the head; she picked herself almost constantly, and at times straightened out suddenly and screamed at the top of her voice, as if in pain. She is resistive to almost all the attentions of the attendants and quite untidy. She took her food only on persuasion, from the spoon.

On June 8 she was found in great *perplexed and helpless agitation*, very much emaciated, with a facial expression reminding one of a miniature of Rameses' mummy, the angles of the mouth pulled down, the profile almost cutting, the forehead occasionally in a play of contractions in efforts to open the usually but half-open eyes, accompanied by even further downward movements of the angle of the mouth, or in vain efforts to say something with pointed lips; the eyes and mouth are red and slightly excoriated from the continual palpation with the fingers and the rubbing; the olecranon region and the wrists and the chest are scaly and almost copper-coloured from the friction in the continual movements of the hands, which go on fumbling tremulously, often with lifted elbows, digging with the index on the bedding or the face or the chest, or pulling the sheets a little. The legs are pulled up and held rather firmly, but the resistance is overcome with some effort. The plantar reflex is prompt on both sides; on the right the toes go down a little and then slightly up, and on the left they all go downward. The knee-jerks are somewhat exaggerated; no ankle clonus is elicited. The patient had been too weak to get out of bed for over a week; twice she had been seen to break down suddenly when she had walked into the hall.

The patient appears to notice the physicians and that they speak to her, since she looks up at once when spoken to. In her fumbling and grimacing she makes vain efforts to speak. "Show me the tongue." She opens the mouth and puts her finger in tremulously. Another time the request caused her to open the mouth and show the lips; but she does not protrude the tongue. She takes hold of a watch held out to her, in a very tremulous and awkward way, and mumbles "Trois heures" and "mo-mo-mo." It was 9.12 a.m.

The examination of the internal organs is very difficult owing to the unrest, the irregular and at times tumultuous breathing, and the frequent and sudden contractions of the pectoral and clavicular muscles. No dulness or râles are discovered. The respiration varies from 20 to 25 per minute when the patient is calm, the pulse between 100 and 110, the temperature 101·8°; the sleep is much reduced; food is often refused. The *diarrhœa* was not constant, as she would several times go two days without a stool, though at other times she would have from two to four stools a day, generally during the afternoon, from 3 to 8 p.m. The tympanitic condition of the abdomen has disappeared. On June 8 she began to refuse the food, pointing to the throat, making a motion as if it hurt her or as if she could not swallow. She took the food into her mouth, but would be unable to swallow it. She shook the head when more was offered her. The sleep became more impaired, and the general condition weaker.

On June 9, at 5 p.m., the patient showed signs of collapse, the fever had risen to 104°. She stared intently at the ceiling, reached up her hands as if attempting to grasp something, mumbling continually. Death occurred at 11.45 p.m.

The treatment had been mainly palliative (against the diarrhœa) and trying to effect rest and better nutrition. The diagnosis first was anxious melancholia, although the depression gradually gave way to a more or less anxious perplexity. The diagnosis of a "central neuritis" became very probable when a typhoid infection could be excluded, and the agitated weakness gave signs of jactation. The *autopsy* was made twelve hours after death by Dr. Dunlap.

The body is that of a small *emaciated* woman. Rigor mortis complete. Hypostasis marked over posterior part of thorax and buttocks. Panniculus adiposus 1 mm., pale yellow. Intestines but moderately injected, filled with gas, moist and free. Omentum retracted, containing very little fat. Appendix normal. Liver not visible anywhere below the rib margin. In the pelvis about 10 cc. clear fluid. Diaphragm at the fourth interspace on both sides. The lungs retract considerably. No pleuritic adhesions.

Pericardium free, with 8 cc. of clear fluid. The left heart fairly firm, the right lax. The pulmonary artery contains a soft mixed clot.

Heart, 135 grammes.—The right auricle contains a small amount of non-adherent mixed clot. The right ventricle is pale, the walls from 1½ to 3 mm. The tricuspid valves are thin and normal, also the pulmonary valves. The left auricle is free. The mitral ring admits two fingers easily. The left ventricle walls are from 8 to 13 mm. thick, pale, brownish-red; the valve curtains are normal.

Left lung, 265 grammes.—The upper lobe gray with a purplish tint. The lower lobe a little firmer and a darker purple. A few scattered pin-head sized *sub-pleural ecchymoses*. The consistency is soft throughout, a little boggy in the dependent parts.

Section: Grayish-pink in the upper, red in the lower lobe. The cut surface of both lobes is smooth and from each a *bloody fluid* and *some froth* can be expressed; much more abundantly in the lower lobe. The bronchi contain frothy pink mucus. The larger vessels are free, except for a few soft dark clots.

Right lung, 300 grammes.—The middle lobe and anterior part of the upper lobe gray and soft. Lower lobe and posterior half of the upper, purplish pink with projecting light pink lobules and on the outer surface of the *lower lobe, three dark red, almost black*, elevated resistant *nodules* about 8 by 8 mm.

Section: Upper and middle lobes in gray portion normal and dry. Lower lobe and posterior part of upper, red with smooth surface; considerable *frothy pink fluid* flows from the upper lobe, practically none from the lower, from which, however, a bloody fluid with fine froth can be expressed. Section through the dark areas shows the nodules to extend about 8 mm. below the surface.

Spleen, 110 grammes.—Flabby, with considerable pulp. Trabeculæ very indistinct. Substance on section quite friable.

Left kidney, 95 grammes.—Capsule splits considerably without tearing the cortex. Surface pale and smooth. One *yellow speck* 1 mm. in diameter. Cortex 3 to 4 mm., pale, with yellowish streaks. The glomeruli appear as translucent points.

Right kidney, 115 grammes.—The capsule tears the substance considerably. External appearance, section and glomeruli similar to left kidney.

Liver, 1,080 grammes.—Smooth, reddish-purple. Section: light purple, with very indistinct lobules. One gall stone the size of a large cherry.

Intestines.—About 13 cm. from the ileo-cæcal valve, in the ileum, is found a shallow ulcer with rather indefinite edges and slightly elevated peripheral zone (about 3 mm. in width). Part of its base has a translucent, healed appearance. The whole depressed area is 16 by 4 mm., the deeper area with ragged base 8 by 1½ mm. No enlargement of Peyer's patches; the mesenteric glands average about 6 mm. in diameter.

Skull, 373 grammes.—Symmetrical, smooth; sutures indistinct. Sulci shallow. Considerable diploe throughout. Slight pacchionian depressions along the sagittal suture.

Brain, 1,100 grammes.—Rather flabby. Lobes separate easily. Pia of convexity thin with but little fluid in the sulci. Cysterna clear. Vessels of base thin. No granulations of the ventricles.

Anatomical Summary.

Emaciation. Small erosive ulcer in ileum. Ecchymoses in one and slight œdema in both lungs. Dark nodules (and beginning pneumonia?) in the right lower lobe. Gall stone.

Microscopically the lower lobe of the left lung contains intra-alveolar clumps of streptococci with barely beginning reaction in the tissue. A small dark nodule shows hæmorrhagic and fibrinous infiltration of the alveoli with but few traces of cocci.

The heart muscle shows beginning fragmentation. Liver and kidney had no characteristic lesion. The intestinal erosion shows hardly any granulations.

When the *axonal reaction of all the Betz-cells* was established, the brain was subjected to an extensive study with the *osmic acid reaction.* The *right hemisphere* yielded:

(1) Sections through the radiations of the frontal lobe as far back as the foot of the central fissure (fig. 3, *a*, and fig. 4, *a*).

(2) Sections through the radiations of the upper part of the motor area and the upper parietal lobule (fig. 3, *b*, and fig. 4, *b*).

(3) Sections through the auditory radication (fig. 3, *c*).

(4) Sections transversely through the hemisphere, at the level of *d* (fig. 3 and fig. 4).

(5) Transverse sections through the occipital lobe.

The *left hemisphere* was trimmed for a series of transverse sections of the basal ganglia, and the somewhat defective results of the study of the right occipital lobe called for a study of a series of sections through the left occipital lobe. The rest of the *brain-stem* was examined in a section through the mid-brain, and several transverse sections through the hind-brain, in the trigeminal segment, the pneumogastric-hypoglossal segment, and the first cervical segment.

The result is recorded in the drawings, and is here summed up as follows:—

(I.) The segmental elements, as far as examined, were little affected. The motor cells of the hypoglossal, the spinal-accessory and vagus, and the facial nuclei, show varying degrees of irregular rarefication of the stainable portions and in each section from two to four cells, with typical axonal alteration in each nucleus. The same holds for the first cervical segment. Only few root-fibres are affected. Among the cells of the nucleus reticularis, in the auditory-facial-abducens segment, there are many with axonal alteration, but a small number perfectly normal. Many cells of the lateral nucleus have the axonal change. The respiration-bundle has but few large lumps of decaying myelin, and the caudal root of the fifth nerve is practically free. In the Gasserian ganglia the cells show moderate shrinkage or none. They vary in diameter between 25 and 60 μ. The free marginal border is practically absent. The preponderating cell-type is that with a marginal zone of rather large stainable lumps, gradually decreasing in number and size until a free perinuclear zone is reached; in a relatively large number of cells the stainable portions are practically limited to the marginal ring, and in about an equal number the medium-sized lumps are rather evenly

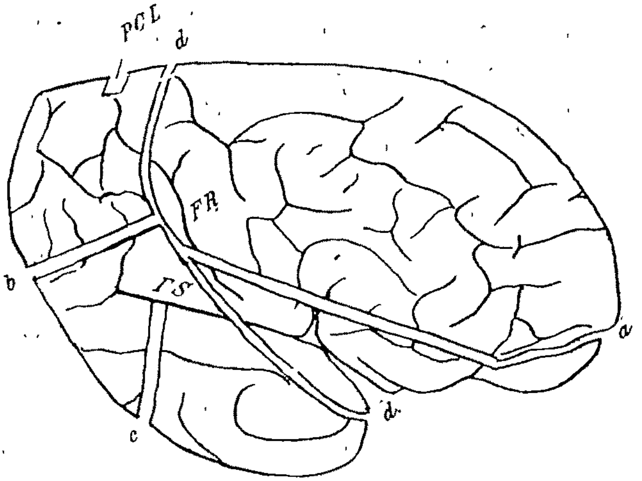

FIG. 3.

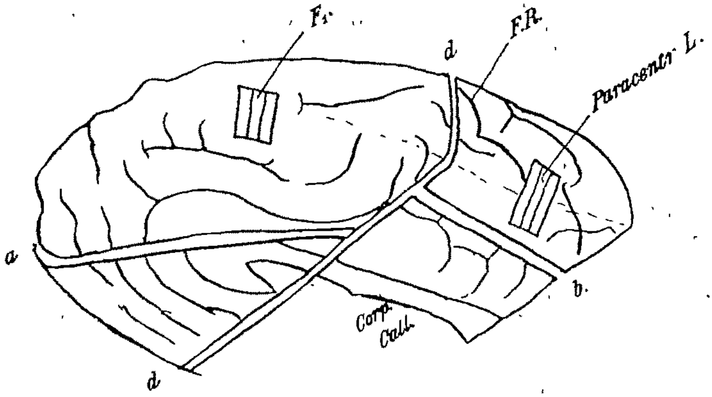

FIG. 4.

distributed from the periphery to the nucleus. The nucleus is normally fairly central and only in very few cells broken through the marginal ring of stainable portions, so that an undoubted *axonal reaction* could be established. It has usually a plain membrane, a diameter of 11-15 μ, a nucleolus of 3·5-5 μ with frequently plain apposition bodies, whereas the sap and linin-frame remain free of other blue granules in the Unna specimens. There are but few "fatigue-nuclei." Moderate pigmentation, usually perinuclear. The sympathetic ganglia present cells of the most varying types, from the rested cell, with even distribution of the stainable substance and clear central nucleus, to those with extensive dissolution of the stainable substance, and eccentricity of the frequently darkened nucleus, even beyond the marginal ring of granules.

(II.) The fore-brain and its dependent parts :—

(1) The *fillet* is slightly affected from the nuclei of Goll and Burdach (many of whose cells show the axonal reaction) to the ventral nucleus of the thalamus, and to the posterior corpora quadrigemina. The *optic tracts* are not affected.

(2) The *thalamo-cortical connections*, as seen in sections through the basal ganglia, are intensely involved to the following extent :—

(*a*) The radiations *between the lateral nucleus of the thalamus and the motor cortex* are very deeply blackened (see fig. 9).

(*b*) The *optic radiation appears* to be free in the external geniculate bodies.

(*c*) The *auditory radiations* have a small number of degenerating fibres.

(3) The *medulla of the convolutions and the centrum ovale*:

(*a*) The *motor region* and its projection fibres are profoundly affected. A few of the blackened fibres penetrate the polymorphous layer of the cortex. The presence of degenerating fibres is not absolutely limited to the central convolutions but overwhelmingly so ; backward the degenerating fibres are sharply limited to the posterior central gyrus. Degenerating fibres to the corpus callosum are not very numerous. From the irregular course of some of the fibres one might well infer that also association-fibres are involved.

(*b*) The white matter underneath *the first temporal gyrus* and the transverse temporal gyrus alone have degenerating fibres, especially the former, while the second temporal gyrus is devoid of them (see fig. 7).

(*c*) In the *occipital lobe* the maximum degeneration involves the forceps, and it is easy to follow into the splenium of the corpus callosum on the one hand and into the cortex on the other, especially into the lateral cortex (see figs. 15 and 16). The optic radiation is relatively free, and also the inferior longitudinal fasciculus. Whether the degenerating fibres of the subcortical layer of the calcarine cortex belong to the forceps, is not definitely settled.

(4) The *cerebral efferent elements* have suffered to the following extent :

(*a*) The *pyramidal tract* shows a marked decay of myelin-sheaths of some of its fibres. The relative number is smaller than the appearance of the cortex would make one suspect, though the difference is not as marked as in case 6. This discrepancy led to a study of the thalamus which shows that so many of the degenerating fibres were thalamo-cortical.

(*b*) The cerebro-pontile fibres are only very slightly involved, so that Türck's bundle as well as the median part of the crus appear free.

(*c*) The fornix is free, with the exception of one or two fibres in a section.

(*d*) The corpus callosum has fairly numerous degenerating fibres in the splenium and about the region belonging to the motor area, but not very clearly limited.

The *typical axonal alteration* was found to be present in the paracentral lobule in all the Betz-cells and in many of the large pyramids. In the frontal lobe the alteration was much rarer throughout the cerebral cortex, the cells are very pale, with great reduction of the stainable substance but practically no diffuse stain of the non-stainable parts. The *blood-vessels* are nowhere infiltrated or thickened, but slightly distended.

III. The *cerebellar apparatus* is involved in three points :

(1) There is a distinct degeneration of a number of fibres in the restiform body in its course around the dentate nucleus to the vermis.

(2) Many of the cells of the dentate nucleus show a beginning of axonal alteration.

(3) The cells of the roof-nucleus have pronounced axonal alteration. The Purkinje cells are free.

Case 8 (from Dr. Budd's service).—*H. B. P., aged* 56. *Alcoholic senile deterioration with stuporous delirious episode. Collapse.*

The father of the patient had a shock at 68 and was mentally feeble afterwards, and died of a second shock. The two brothers and three sisters are free of hereditary features.

The patient was born in Maine, 1844. He went to the war at 17 and left the service in a year with "black measles and dysentery." "He did not recover his health for many years." He worked on the farm two or three years, and then became lineman on telegraph-lines, a good worker with highest wages. In this work he sustained *several injuries* from falls; he also was given to alcoholics and venereal excesses (many drinks of raw whisky every day; no venereal infection known of). He married and had children; about 1890 he left wife and family on account of his drinking and abusive and hot-headed disposition, and since then he has boarded in different places.

November 10, 1898, he was run over by an express-waggon. He was admitted in the Boston City Hospital with lacerated wounds of the face, and the next day he developed a probably *alcoholic delirium* which lasted nearly a week and necessitated the use of a straight-jacket. He was discharged well November 26, but his daughter thinks that he began to fail after this. He drank more, "seemed to stay half full all the time," and it happened that when he took down a wire for repair he went away forgetting to put it up again. He had to be given the easiest work, and about February he had to give up even that, because he was unable to go to the shop on account of physical weakness, especially in his legs. He spoke of getting a pension from the government and was very impatient about it.

For the last month he wandered about the house acting like a child, but up to within a week before his commitment he talked rationally and sensibly and was neither troublesome nor violent; his memory only was very defective.

About a week before his admission to the hospital he began to produce a confused talk about Heaven; that his son was being "buncoed" out of Heaven, that there was a war going on in Heaven, &c. He began to go two to three days without eating, knew those about him, but was very restless. He would be sitting down talking and suddenly jump up and go out into some other room, look around and come back again, saying, "It's all right," then he would sit down, only to get up again in a few minutes. He slept very little at night.

No evidence of delusions of persecution, of reference, of robbery or of expansive ideas, or of hallucinations could be obtained from informants.

Lately he has been very weak but not in bed. The last day at home he was arrested at the instigation of the boarding-house keeper, because they said he grabbed one of the children by the throat. The daughter says he was not cross and irritable, and thinks he really intended no violence. He also threatened and tried to jump out of the second storey window.

On admission, May 19, 1900, the patient required assistance to get into the ward on account of his apparent weakness. On the evening of the 19th and the morning of the 20th, he had no idea of the city or nature of the place, but refused to assent to absurdities (stable, &c.). He twice said he came from Omaha, Nebraska. He did not know the year, month or day, and when pressed for the season, said it was winter. He was told the place, but on the evening round of the 20th he did not remember being told, and he had no better orientation, except that he said that the doctor "acted like a doctor," because he felt his pulse. He did not remember seeing him before in the morning, but said he had seen him before he came here. He constantly said there was nothing the matter with his mind. On admission his pulse was very irregular, intermittent, dropping one every two or three beats.

Physical Examination (May 22).

The patient is found to be a tall, moderately slender, well developed and well nourished man with thin gray hair and beard, abundant growth of hair over the thorax and abdomen, and dark, tanned complexion. Skin somewhat flabby, warm, moist and free from eruptions. Recent abrasion on lower third of right shin, 2 by 4 inches. Over left malar-bone, a scar 2 cm. in length. No other abrasions or noticeable scars. The left *clavicle* at junction of middle and outer thirds has an old callus, the inner end overriding the outer; union is complete. *Skull* symmetrical; *adherent lobules;* palate of medium height and width; *teeth* sound but well worn. Hands well formed. Large toe of each foot is much abducted, and the *metatarso-phalangeal joint is very prominent.* Penis and testicles normal; no scar is found.

General condition.

He remains in bed, but now and then gets up and goes to the closet; does not complain unless asked, then saying nothing further than that he "is sick" (ill). His tongue is clean; appetite only fair; bowels regular; sleep only by aid of hypnotic.

Eyes.—Dark blue, well opened and prominent. Conjunctiva clear: *pupils slightly displaced toward inner side, but* *equal*, 2½ mm., react to light and accommodation. Movements unrestricted. Vision normal. No hemianopsia.

Smell unaffected, and *taste* unaffected.

Hearing.—Good on either side.

Cutaneous sensibilities.—Touch and localisation, prompt and accurate. Pain and temperature tests are unreliable because of resistiveness of patient; believed to be normal.

Motor functions.—Muscular power of arms, equal but poor. Power of legs equal and good. *Gait, slow and feeble. After standing a few minutes he begins to reel as if dizzy, and complains of weakness, and he is helped to bed. Very slight individual tremor of fingers, becoming more marked on rotation. Tongue small and median, with jelly-like tremor at sides. Marked swaying in Romberg position.* No speech defect.

Reflexes.—On plantar stimulation the smaller toes flex somewhat, the large toe remains still. No ankle clonus. Achilles reflex, equal and moderate. *Knee-jerks both exaggerated, perhaps a little more so on the right. Elbow and wrist-jerks are equal and somewhat exaggerated. Cremasterics slow and long continued.* Epigastric not obtained.

Lungs.—Expansion equal and good. On percussion at the right apex, in front and over the entire surface of the lower lobe behind as far as the angle of the scapula, there is a higher pitched resonance. The respiratory murmur is relatively somewhat harsher over this area, and the expiration slightly prolonged. No râles are heard; no increase of fremitus.

Heart.—Pulse 64, *intermittent and irregular*, but full and of good quality. *Radials thickened, brachials slightly thickened.* Temporals not visible. Apex beat not visible, but felt weakly in the fifth interspace, inside of nipple line. *Heart boundaries.*—12 cm. to the left of median line at the sixth rib, 4 cm. to right of median line; whole width 16 cm. There is a distinct systolic murmur, rather rough in quality, best heard over the sternum, opposite the attachment of the third ribs, and better over the aorta than over the pulmonary ostium. No marked accentuation of the second sounds.

Abdomen.—Stomach even with costal margin. Liver from the sixth rib to the costal margin. Spleen not palpable. Inguinal glands felt plainly.

Urine.—May 21. Dark amber colour, clear with light cloudy sediment. Acid 1030, no albumen or sugar. Calcium oxalate, amorphous urates, epithelial cells, no casts.

May 23. Asked where his home is, he hesitates a long time, and finally says: "Have none." "Where are your people?" "Have none." "Are you married?" "No." "Where did you live before you came here?" "Don't know."

Mental Status. (*May* 29).

Attitude and manner: Confined to his bed, he tries to run out or to pull clock down; At times he is irritable; feeds himself and is cleanly; often calls out.

Too weak to stand more than a few minutes at a time, he had been in bed since admission. At first he seemed to be ill at ease in bed, and often tried to get out, a number of times succeeding. He would then run to the door and try the handle, but on finding it locked, he would turn away saying nothing. Once he ran and reached and tried to pull the clock down from the wall and was prevented just in time. He would give no rational answer to questions about his conduct. He usually lies flat on his back. He often called out when lying in bed, saying such words as "whoa," "Over in the ten-acre lot," "Her name is Bailey," "All right," "It looks like it," "What?" "There, there," "Yes, there, east corner, 40 rods—timber land," &c., and then relapses into silence for some time. He shows but little interest in his surroundings, and answers questions in a low tone. During the first few days he showed considerable irritability when restrained from getting out of bed, but after a few days he did not try to get up so often and only showed occasional temper. Even when angry he said very little. He has been uniformly clean, always asking for assistance to go to the closet, or even to expectorate, and he has fed himself without trouble. On occasions he will absolutely refuse to eat one or two meals, and then again will drink milk when he does not take solid food. Once in a while he pulls and picks at the bed-clothes a little.

The following gives an idea of his stream of thought under a minimum of urging:—

Conversation: Volunteers rambling remarks, but finishes sentences.—"I've heard nothing from the general report. I think it is proper and right. I haven't been to the meetings only once." "Eh!" "Yes, that's all." "What have you got to say?" "I've been telling you about going into it. I suppose it was fixed over long time ago before I had anything to do with it." "What?" "What collects the seed?" "What do you mean?" "I don't know. I suppose you belong to the government, and know the seed of the womb. I suppose it would proper, you ought to know." "What else?" "Why, I've got to reckon everything right through. When they are full they will take no more, that is all there is to that." "What is that?" "That's so, they give birth to young." "Who?" "A hive of bees isn't it? It is made by bees. They suck the seed from different meals and far different climes and different places." "What seed?' "The seed to manufacture the substance, its a honey." "Who spoke of honey?" "I don't know. That is the way I've always heard." "What have you been talking about?" "What have I? I don't know. I've been talking but little on account of"—(stops). "What?" "I didn't say anything."

Orientation: He calls the doctor as such; says it is 1900, *but does not know place, day or month; his answers show confusion.*—"What place is this?" "Wakefield Junction, is it?" "No." "I don't know." "What building is it?" "I couldn't give the first guess." "Why do you think it is Wakefield?" "Well, I don't know. I surmised, that's all." "What day is it?" "You have me now." Question repeated. "I don't know. I couldn't say." "What month?" "July." "Why do you think so?" "That will have to go. I guess you can put it down or not, just as you choose. They don't interest me very much now." "What year is it?" "Well, some say it is one, some another. It is supposed to be 1900 by some." "What do you say?" "Well, I don't know." "Is this your home?" "I wasn't born here, no." Question repeated. "Well, I come here before I was married. I had children before I enlisted." "Do you know me?" "Doctor, yes. Dr. Jones," (rising inflection). "Are you sure?" "Well, I heard you called that a good many times. I don't know as I was ever introduced to you, but I know you attended me, that's all." "When?" "Well, ever since they transferred the old Grand Army out, and transferred the old pensions on to this list to be transferred right on." "What was the trouble?" "Well, disability. I've been broken down pretty bad. You've been with me and ought to know pretty well."

Memory for remote events: generally fair, specifically poor.—"Where were you born?" "New Gloucester, Maine." "When?" "About 55 years ago." "What date?" "October 16. I was born in 1844." "Did you go to school?" "I was four years old." "How long did you go?" "I didn't go but little; went summers and winters three months each." "How long?" "I had to work the farm summers, and could only go winters." "How old were you when you stopped?" "17, I think." "What did you do then?" "I worked in a cotton factory in

Lewiston." "How long?" "Pretty near a year." "What then?" "Enlisted in the army. I wasn't there very long. I was ill considerable." "In any battles?" "Yes, I was in the seven-days' battle before Richmond." "Who was your general?" "General McClellan at that time." "What regiment were you in?" "The 11th Maine." "What date were you discharged?" "I haven't just the date." "About?" "I couldn't tell." "What year?" "I don't know." "How long were you in the war?" "You can reckon that up yourself as well as I, or better." "What year was it when you quit the army?" "It was '63, I guess, I couldn't say." "When were you married?" "Tenth day of January, 18—1880." "What?" "Eh?" "When were married?" "I couldn't tell you the day and date." "When was it?" "I don't know. I knew the day and date at the time, but I don't now." "How old were you?" "I don't know. I suppose I was 17 or 19, something like that—19 I guess." "What was your wife's name?" "E. D." (Correct). "Where is she now?" "I don't know." "Is she alive?" "She was, the last I heard of her." "Where?" "M———" (Correct). "How long ago?" "Some time ago." "Ten years?" "Oh, no." "One year?" "I've seen her in a year." "Where?" "At M———" (Correct). "Any children?" "Yes, three girls and one boy." "Where?" "The girls are all married; the boy lives with the other ones. All the children live together, except the one, Edith. She is in the same place, but different, in the same town, married." "What is your business?" "I've been working at electrical work considerably, called a lineman."

Recent memory: very variable, confused answers at times; stereotyped: "I don't know."—"How long have you been here?" "I don't know." Question repeated. "I don't know as I've been here but once." Question repeated. "Not knowing, I couldn't say, otherwise, don't remember." "A month?" "I don't know." "A year?" "Couldn't say." "When did you see me last?" "I saw you when you put those—heard the remark you made to—heard it before." "When?" "You came through here and put those pamphlets on my bed to-day." "What remark did I make?" "You said: 'Oh, yes, I prefer being on this side,' and you skipped over." "What did I mean?" "I suppose you thought it was the last day and the world was coming to an end, or an earthquake or something. A lot of foolish stuff. It sounded foolish." "Where did you come from?" "I don't know." "Why did they bring you?" "Who brought me?" "Don't you know?" "No, I don't know, I'm sure." "Where did you work last?" "For Webb, I believe." "Where?" "I suppose it was here." "Where?" "Wakefield Junction, is it? I don't known where."

Grasp of surroundings exceedingly poor.—"What kind of a place is this?" "An armory, I suppose." "Why are you in bed?" "You said I was sick." "Are you?" "I ain't very well." "What is the trouble?" "I told you I was all broken up, and you said here was a tender spot, and you ought to know, I suppose. If you call me a healthy man, let it go at that, and I won't say anything." "Do you know anyone here?" "No." "Did you ever see them before?" "What, the people that come here to view the——? no, I don't know." "What time is it?" "I don't know" (looks out of the window), "I don't know. I should say it was considerably after noon." "What time do you have supper?" "Well, different people have supper at different times." "What time do you?" "Sometimes one and sometimes another; sometimes I don't have any." "Ordinarily what time?" "Well, split the difference between that and—and you will get it as near as I can tell you. Ordinarily I get what I want to eat." "What time do you see me in the morning?" "Eight o'clock, is it?" "How many times have you seen me to-day?" "Three times, I guess." (Right.) Asked what the attendant did, he said: "As regards the collection of the seed, he is in it all right." Reads a watch correctly. "What am I writing?" "I think you are writing a full set of—I don't know—to have the thing progress, I guess." "What purpose?" "They have it to drill with, I suppose."

General information: (Answers not reliable).—"Who is president?" "What day?" "Now." "When now?" "1900?" "Do you know?" "No." "Who was shot during the war?" "I don't know." "What president got shot?" "I know a man who got shot, but no president."

Calculation: poor for combinations.—$3 + 9$? "27." $4 + 4\frac{1}{2}$? "12." $9 + 9$? "81." 13 and 7? "19." $2 + 13$? "26." $9 + 12$? "108." $3 + 13$? "I don't know." 17—9? Pause. "*It would be* 9."

Counting.—He counts from 1 to 20 in five seconds, keeping on up to 50 rapidly.

Hallucinations: are not elicited, although he often calls out (see above).—There has never been any direct evidence of hallucinations, and nothing of that character can be elicited by questioning the patient.

Delusions: nothing definite elicited.—"Did you have trouble with your wife?" "Yes." "What?" "A little domestic trouble." "What was it?" "Just a little." "What?" "Her daughter had a kid to-day." "Is that the trouble?" "Yes." "Have you any property?" "No." "Do they treat you well here?" "I've been treated all right." He occasionally jumps out of bed and tries the outside door, but never gives a rational explanation.

Insight: perhaps partial; answers contradictory and lacking animation.—"Is there anything the trouble?" "I have heart trouble bad." "For how long?" "Five or six years. Had it pretty much all my life." "Has your mind ever been affected?" "Some, not much." "What caused it?" "Liquor caused it, I presume." "How did it affect you?" "When I didn't have any it affected me." "How?" "Because I wanted it." "Did it affect your mind?" "No, it didn't affect my mind any." "Is it affected now?" "It isn't any better than it ought to be." "Have you ever been crazy?" "No." "Were you ever out of your head?" "Yes." "When?" "I was up to the City Hospital awhile." "What was the trouble?" "I got injured by a team." "Are you as sound in your mind as ever?" "You are the best judge of that." "Are you confused?" "Yes." "When?" "Well, most of the time" (in a lifeless manner). "This is an insane hospital, why were you brought here?" "Because of that arm." "Have you ever been insane?" "I told you no."

Reaction to questions: monotonous, forgetful of questions, occasionally irritable; some surprisingly accurate answers.—He lies in bed, looking out in a calm, deliberate, rather sleepy way, occasionally glancing at the speaker with a little gleam of animation, answering the questions in a low, monotonous tone, but when asked to repeat always repeating the same word or phrase, and finishing his sentences. Sometimes he makes an irrelevant or confused answer. This is more noticeable where he is persuaded to talk voluntarily (see conversation). When direct questions are put, he often makes a surprisingly accurate reply, but will perhaps make an equally foolish answer to the next question. Now and then when a question has been repeated several times, he will show irritation and elevate his tone somewhat to emphasize his reply, or sometimes saying "I told you before," or "I don't know, I tell you," then readily relapsing into his easy way again, in order to keep it in his mind.

June 16. *Restless; out of bed; diarrhœa; sudden death with cyanosis.*—Patient continued to remain in bed, observing in listless manner those about him; occasionally speaking as if to himself as already noted. He was less restless than when admitted, but would occasionally try to get out of bed, and seemed especially restless at night, so that quite often he required light restraint. He at first showed no mental change either in voluntary speech or in reaction to questions from that already recorded. About two weeks before his death he was found to have fallen from the seat in the water-closet and to have bruised his forehead. He was lying quietly on the floor, and was not unconscious or more than usually stupid. Previous to this he had been taken to the closet after he had voluntarily started, and was always able to sit alone and to walk back with assistance. After this, however, he seemed to be growing weaker. He would often try to get out of bed, and when asked if he desired to go to the closet, he would say "yes," but he was not able to walk the whole distance, and unless he was helped he would soon sink to the floor. He was thereafter carried until about one week before he died, when he was not allowed to attempt to walk. He became noticeably weaker during the last week, and slightly more restless during the day, often pulling the bed-clothes about and attempting to get up, but when prevented he would lie back and apparently sleep. On several occasions when he succeeded in getting out on the floor, he was only able to walk a short distance, and would then sink easily to the floor, apparently too weak to go farther. There was at no time any evidence of muscular twitchings or spasms, nor any drawing of the facial muscles. He gradually became less talkative and observant. When spoken to he answered in a low tone, and generally irrelevantly. When his answers were relevant, it was thought he did not understand, but that it was merely accidental.. He often took no notice of questions at this time. It was observed some two or three weeks before death that he would hold saliva in his mouth for some time waiting for help from the attendant, as he would readily expectorate when given opportunity. His expression was stolid and heavy. He often spoke about "electricity" and "wires," but not in connection with anything about the wards.

About four days before death he began to have diarrhœa, and he then was so stupid that he did not appear to notice when his bed was soiled. He had to be fed by a spoon, and passed the day lying flat on his back with eyes closed, and could hardly be aroused. He would occasionally rouse up slightly, but only for a few moments. He had not attempted to get out of bed for some time before the time of death. The condition of the reflexes did not change.

He died June 16, in the morning, about 7 o'clock. After getting out of bed and walking through the door, he sank to the floor easily, fell backward in a heap, began to gasp in a strangling way, and to roll his eyes back. He was placed on the bed, and only breathed a few times afterward. The attendant noticed that he was at first very cyanotic over the most of the body, but, shortly after, this faded away.

Autopsy, June 16, at 11 a.m. Body of a well-developed and fairly-nourished man. Body heat present. Rigor in jaw, slight in legs, little if any in arms. Hypostasis considerable in dependent parts, purplish lividities on thighs and legs, even on anterior portion. Scrotum slightly abraded on right, lower half dark purple. Panniculus, about 2 cm., rather pale yellow. Intestines moist, but slightly injected; omentum retracted. Mesenteric glands not enlarged. *In the mesentery* of the ileum, near the scrotum, are about a half-dozen *calcareous nodules*, varying from small pin-head size to that of a small bean. Some of these lie near the intestine, others at the mesenteric attachment. Appendix normal. Diaphragm, fourth rib on right, fifth interspace on left. Liver in mammary line about 4 cm. above costal margin, nowhere visible on opening the abdomen. Lungs retract considerably; at the *right apex there are two narrow bands of strong adhesions*. Elsewhere the lungs are free; very little fluid. Pericardium contains about 10 cc. clear serum; pulmonary contains soft clot; right heart flabby and full of blood, left firmly contracted.

Heart, 315 grammes. Over the anterior surface of the right ventricle is a *thin white patch* (milk-spot), 2 by 3 cm.; *coronaries are tortuous, thickened and roughened*, embedded in a small amount of fat. *Over the left ventricle are two groups of pin-point to slightly larger ecchymoses.* Right auricle contains a dark, rather soft clot mixed with lighter clot. This does not adhere to the endocardium. Appendix contains a tough clot between its trabeculæ. *Tricuspid opening admits easily five fingers* (perimeter, 14 cm.). *Right ventricle enlarged;* contains a stringy, light clot; walls from about 1 to 3 mm. Colour rather pale. The tricuspid valve curtains are thin and translucent. *Pulmonary valves are large, flabby, thin* and free; the measured diameter of the posterior valve is 4 cm. Left auricle free, except for slight stringy, non-adherent clot in the auricular appendix. Mitral ring admits two finger-tips rather easily. Left ventricle walls are rather pale brownish, from 2·5 to 20 mm. *Mitral* thin and normal, except for a *slight yellow streak*, a little elevated and a little roughened on the posterior surface. Aortic valves leak water slowly; thin and translucent in general, with but slight thickening near the attached margin of the right posterior valve. *The ring and beginning of aorta show small streaked yellow elevations, varying* from pin-point to 3 mm. in diameter.

Left lung, 235 grammes. Dark steel-gray, soft and uniform; interlobular fissure absent in its middle third, without any evidence of adhesions. On section, tissue is grayish-pink, dry and leathery. Small amount of dark blood is squeezed from the vessels. Bronchi very slightly injected.

Right lung, 290 grammes. Externally the same as left, except for a *patch of thickened pleura* about 3 by 1 cm. at apex, and a few emphysematous-looking lobules near this patch. Middle lobe faintly indicated by a depressed line near the anterior margin, and by a notch, 2 cm. in depth, at the fissure. Section similar to left, except for the upper lobe, which is of a pale gray.

Spleen, 155 grammes. Purple; little pulp can be scraped off; trabeculæ fairly distinct, substance rather friable.

Kidneys, left, 155 grammes. Surrounded by a thick layer of fat; substance *deep purple*. The line of lobulation shows very plainly, and the capsule splits considerably and is very thin, it tears the substance slightly. Section very dark purple. Glomeruli give a granular appearance

Right kidney, 160 grammes. Similar to left.

Liver, 1,645 grammes. Normal in external appearance. Section rather light reddish-brown. Lobules very indefinite.

Skull-cap. Fairly regular, rather long; sutures well preserved; sulci moderately deep, in part ragged; very little diploë; marked patch of depressions about 15 mm. outside of median line, corresponding to Pacchionian granulations, visible on the outside of the dura. Longitudinal sinus practically empty, with broad fields of Pacchionian granulations. *Dura moderately adherent* to skull in frontal region. *The falx has a great number of perforations*. The inside of the dura is free; Pacchionian patches correspond to the entrance of the largest veins into the sinus. *The pia of convexity is slightly hazy, very œdematous*. The blood-vessels of the base are thin and tender, except the entrance portion of each middle cerebral. The *cisterna is thin, with a few flaky thickenings*. The lobes are very easily separated. *Weight* of the *brain in toto*, 1,375 grammes. The fourth ventricle is not granular.

Anatomical Summary.

Dilatation of the right ventricle, the tricuspid, and pulmonary rings. Congestion of kidneys, spleen, and probably liver. Ecchymoses on surface of left ventricle. Œdema and slight haziness of pia of convexity; cisterna shows few flaky thickenings. Moderately adherent dura. Perforations of the falx cerebri. Arterio-sclerosis of coronaries, aortic ring and beginning aorta; slight of one mitral flap; very slight of aortic valves. Milk-spot on right ventricle. Narrow bands of adhesion and thickened pleura at right apex. Calcareous nodules in mesentery.

The *paracentral lobules* show very strikingly the pure axonal reaction of practically all Betz-cells and some of the larger pyramids without disturbance in the general appearance of the cortical tissue. Many of the large cells resemble considerably the vortex type of fig. 2. In the frontal cortex many cells show a similar alteration—rarefication of the stainable portions of the cell-body and dendrites, and occasionally a cell with fairly typical axonal alteration (fig. 2a).

The medulla has extensive axonal alteration in almost all the cell types, except the hypoglossal cells, which differ but little from the average appearance in beginning senescence. Many of the cells of the nuclei of the posterior columns, especially those of the external nucleus of Burdach and also those of the lateral nucleus, are markedly affected. In the cerebellum the roof nucleus and the dentate nucleus have the axonal alteration in practically every cell, whereas the Purkinje cells are not affected.

The Gasserian ganglia are free; the sympathetic cells show varying types. The *examination with osmic* acid was limited to the basal ganglia of the left hemisphere, the mid-brain, and the hemispheres of the cerebellum. The number of blackened and decaying fibres is small. They are limited to:

(1) The thalamo-cortical tract of the motor and visual areas.

(2) There are few fibres in the red nucleus.

(3) The pyramidal tracts are very little affected, and the same holds for the fillet.

(4) The restiform body is considerably involved.

(5) The corpus callosum has but few degenerating fibres, and these are limited mainly to the splenium and to the motor region.

To these cases *Dr. August Hoch* kindly allows me to add another one accidentally discovered by him. Dr. Hoch's case was admitted to Dr. Charles F. Folsom's service at the Boston City Hospital, November 16, 1898. The patient was a carpenter, according to his own statement 58 years old, born in

Maine, with negative family history. The history is difficult to obtain on account of disinclination of the relatives. Alcoholism is denied.

For about two years the patient has had considerable headache and has been slow in thinking. For a year he has been unable to keep his books, but continued to work until three months before admission. Since then he has had vertigo, swollen ankles, delirium at times (?), was more irritable in his home life, had some abdominal pains, and for two or three months looseness of the bowels, which "pulled him down." He finally began to fall on the street, and one day children had to bring him home. He looked 65 or 70 rather than 58, and was really ill two weeks before he went to the hospital. He would sit on a chair with a cap drawn down over his face, and would not say anything to anyone. He finally became restless, and could not be controlled any longer by his wife. He would pick his clothes, jump over the foot of the bed, and follow his wife. He would "holler murder." His diarrhœa became worse. His wife would no sooner get him cleaned up than he would be all "mussed up again." He could not control it, although he tried to go to the closet. It took him a long time to get down stairs on account of a tendency to fall backward, as he did on the street. He never lost consciousness, and always got up by himself. He did not want to be taken away where his wife could not go with him.

On admission it was noted that the pupils were normal; arcus senilis and irregularity of arterial walls were present, but a regular equal slow pulse. No other abnormalities. November 20.—"Since entrance the patient has had hallucinations; the temperature has been subnormal; the bowels much better. November 24.—The patient is delirious and making strange movements of the hands all the time; does not recognise his wife, and will not answer questions intelligently. November 28.—Marked rigidity of muscles for the past few days; no paralysis evident; he makes peculiar purposeless movements, flourishing the arms about and often rolling fingers and thumb together; continued stupor and delirium.

December 3.—The patient appears weaker each day. The mental condition continues the same; peculiar movements also continue. Flexion of head on chest is resisted; rotation is normal; pressure on the back of the neck and tapping on the head causes apparent pain, even hyperæsthesia. The abdominal walls and the muscles of both extremities are in intermittent tonic spasm and fibrillary twitching. No elbow reflex; knee-jerks sluggish; no patellar clonus. Slight pressure of skin causes pain. Flexure of legs on thigh causes intense pain. The plantar reflexes are increased; the abdominal, cremasteric, and dartos absent. December 6.—The patient has continued about the same, gradually weakening. Tracheal râles developed this morning, and the patient died at 2.30 p.m. The temperature had ranged between 96° and 97°, and only once, December 3, reached 97°. The clinical diagnosis was "Tuberculosis, diarrhœa." The autopsy, made two hours after death, established the existence of lobar pneumonia, adhesive pleurisy, dilatation of the lymphatics of the pleura; the dura was firmly adherent along the longitudinal sinus; the pia normal, the basal vessels absolutely free of arterio-sclerosis. The brain weighed 1,470 grammes, and was firm and macroscopically normal. The appearance of the body was that of a man of 75.

The paracentral lobule and first frontal convolution were examined by Dr. Hoch and Dr. Dunlap with methylin blue, Weigert's mitosis stain, Berkeley's Golgi reaction, Cox-Golgi, and Marchi. The result was the finding of the axonal reaction in the Betz-cells, the presence of a small number of degenerating subcortical fibres, and practical integrity of the tissue as a tissue, so that general paralysis was absolutely excluded.

Similar lesions have been reported, since the note on my first two cases, by Drs. Worcester, Maurice Faure and John Turner. To these are added my eight later cases, and the one of Dr. Hoch. A careful search in the literature of melancholia and other mental disorders brought further to my notice an article by Joseph Wiglesworth, "On the Pathology of Certain Cases of Melancholia Attonita or Acute Dementia, with a report of two cases" (1883). In order to convey a better idea of the material on which my conclusions are to be built, the cases are here reproduced in abstract:—

Dr. W. L. Worcester's case of Landry's paralysis (*Journal of Nervous and Mental Disease*, 1897):—

A widow of 40, during the middle of December, 1895, three years after the menopause, began to be troubled by the delusion that she was pregnant and poisoned from a dead fœtus; she consulted a number of physicians, and finally remained in bed. Later, she read of Gov. G. having died of Bright's disease, and concluded she had it too; indeed, about the middle of March her urine was found to contain albumen and casts, not previously found. She also had numbness in the hands from that date, and on April 9 she became awkward in their use; two days later she appeared paralysed in all the extremities. April 14, there was slight paralysis of the left facial nerve (including the ocular branch); in the extremities the parts near the trunk were more paralysed than the fingers and toes. The knee-jerks and cutaneous reflexes were absent. Diminution of faradic irritability and abolition of the same in the thighs, upper arms, and pectoral muscles, were found. There was great loss of sensibility of the extremities; the head and neck were free; the patient was rather apathetic. The *post-mortem* findings were: axonal reaction of the large cells of the motor cortex; beginning axonal alteration of the motor cells of the cord; practically no degeneration of fibres, neither in the cord nor in the peripheral nerves.

Faure's cases (*Revue Neurologique*, December, 1899) probably do not fit altogether into our picture; they are mainly cases of alcoholic-cachectic polyneuritis or delirium, from a general hospital:—

(1) A woman of 42, with cachexia, cancer of the uterus, hydronephrosis, frequent convulsions, profound mental confusion and typical axonal alteration of a great proportion of the large pyramids, slight sclerosis of the vessels and a slight degree of embryonic infiltration.

(2) A woman of 38, with tuberculosis of lungs and liver; very fatty liver, and queer incoherent and childish behaviour before death. The arrangement of the elements in the cortex is normal; but a small number of the large pyramids show axonal degeneration. No other lesions elsewhere.

(3) A woman of 30, with alcoholic-tubercular polyneuritis with mental confusion (the extent of the lesion is not stated).

(4) A woman of 32, also alcoholic and tubercular, with slight polyneuritis and mental confusion (the extent of the lesion not stated).

(5) A tubercular-alcoholic man of 35, with occupation delirium and a final temperature of 41°. A few cells show axonal reaction.

(6) A woman of 50, with acute lobar pneumonia and systematised deliria. Pigmentation of the cells; in a few the axonal reaction.

Turner's cases (Brain, 1899) are all the more important, since they come from a clinical material similar to mine and because they are accompanied by an excellent description of the clinical manifestations. The anatomical condition was studied by him in the cortex only; hence, probably, his conclusion, expressed by the title of the paper. The cases are:—

(1) A woman of 45, with a history of alcoholism, fits, and melancholia in the family, alcoholic herself, depressed, with violent outbreaks for eighteen minutes; delirious and extremely emaciated. Finally, parotitis and broncho-pneumonia, with jactation of limbs, exaggeration of knee-jerks, absence of plantars, coma.

(2) A widow of 52, following an influenza (in July), became irrational, with alternate periods of depression ("a lost woman") and hilarious excitement; after four months she was excited one day and quiet the next. She had no tremor, but exaggerated knee-jerks. About the eighth month of her illness she began to fall frequently; a week later she was unable to stand, became lost and silly; one day, five weeks later, when excited, she was "rushing about the ward and falling about," and after two months of this motor disorder she died with diarrhœa. The brain weighed 1,198 grammes, and was otherwise normal.

(3) A woman of 42 was admitted in 1895, with a history of eighteen years of delusional insanity (some women were plotting against her and her husband). April, 1898, she developed agitated melancholia, began to fail. In May, her knee-jerks were exaggerated; she was much agitated. In July she had occasional diarrhœa. In September she became demented, dirty, very greedy. In November she lost the use of her legs, fell often, and was restless and bedridden. In December she exhibited involuntary jerking movements of the arms and legs, and risus sardonicus, and succumbed to exhaustion. Atrophy of the internal organs.

(4) A sober woman of 37, always hysterical, was nervous and apprehensive for a year, would not leave the house, lost the use of hands and legs, shook and fell often, and was finally committed with screaming attacks, greasy skin, absence of knee-jerks and plantar reflexes, but equal pupils, and no reaction to pricking. Mostly incoherent ejaculations. Death in four days. Slight atrophy of frontal convolution; brain weight 1,137 grammes. The radial and post-tibial nerves in degeneration.

(5) A woman, born in 1830, who had a three months' attack of melancholia in 1889 and was readmitted in 1890 with deep depression and agitation, but free of motor disorders. January 24, 1899, she took to bed with diphtheria and died February 6,

1899. There was found arterio-sclerosis of the basal arteries, œdema of the pia, atrophy especially of the parietal convolutions and a brain weight of 1,283 grammes. Turner raises the question whether the axonal alteration might not be due to the asthenic type of the diphtheria.

(6) A man of 36 whose father had been insane, was taken with incoherence, violence and raving shortly before admission; was restless, resistive, incoherent, not heeding questions, filthy, and normal physically but for involuntary jerking of the limbs and finally inability to stand. Collapse five days after admission. The meninges were very opaque over the vertex, thick, not adherent; atrophy of the convolutions. Brain weight 1,412 grammes.

(7) A man of 52 whose father had been melancholic and committed suicide, was admitted in 1892 with agitated melancholia which passed into dementia; 1894, weakness of the lower limbs came on; in 1897 he became bedridden, and died in 1898 of phthisis. There was atrophy of the frontal convolutions and dilatation of the lateral ventricles. The brain weighed 1,296 grammes.

Nine further cases published by the same writer are to be found in the *Journal of Mental Science*, vol. xlvi., pp. 719—720. The notes are very brief. It appears that at least case nine of Turner's series fulfils the desirable conditions for a diagnosis of general paralysis. Two cases were imbeciles, at least case 14 one of chronic alcoholism with delirium, and cases 8, 10, 12 and 13 probably some form of melancholia.

Wiglesworth's report contains two cases, one of "melancholia attonita," and one of "acute dementia."

(1) A woman of 48, addicted to gin, developed delusions of persecution eight months before admission, and two months later delusions of poisoning, but appeared normal to friends; one week before admission she took to bed, became weak, mute, shaky when walking; in the second week she was a little brighter but still confused, the third week she did some needlework once or twice, wandered restlessly, asked for the bill. She was feeble, had tremor of the hands, and could not protrude the tongue well. The fourth week she was restless and excited one evening. She was sad at heart, nobody cared for her, she had a child at the top of her head. She rolled on the floor and had much tremor. The sixth week she was alternately dull and talked nervously. The seventh an attack of diarrhœa came on and twitchings of tendons

and sub-febrile temperature; the eighth week the diarrhœa was arrested. The patient had a fixed expression; the arms were kept rigid, and there was occasionally a slight jerking of the tendons. Temperature normal. Death. Typical alteration of many pyramidal cells. Tuberculosis of the left suprarenal capsule.

(2) A woman of 30, married ten years, sterile and of poor health. One week after an attempt of her husband to commit suicide she became much excited, was in a panic, clutching hold of everybody, muttering words like "judgment," &c. She showed occasional tremor and in a week more or less rythmical jerks of the right arm, self-absorption, resistance, and the picture of acute dementia. Death on the nineteenth day from mere exhaustion. Wiglesworth remarks that the cell changes (inflations of nerve cells with dislocation of the nucleus) are probably analogous to those described by Charcot as occurring in the large cell of the anterior cornua of the spinal cord in so-called "infantile paralysis."

To this we add that Spiller and Hirsch have found similar changes, Spiller in the cortex of an imbecile who died with meningeal hæmorrhage (the description only shows the axonal alteration of the Betz cells; there are no Marchi specimens of the cortex, and the material was preserved several days after death), and Hirsch throughout the nervous system of an imbecile, also without Marchi impregnation.

ANALYSIS OF THE AVAILABLE CLINICAL AND ANATOMICAL DATA.

On looking over this material, its heterogeneous clinical character can hardly escape attention. Taking for granted that we have good reasons, owing to the uniform findings at the autopsy, to subject all these cases to an analysis with some expectation of finding common clinical features, we meet with some disappointment at first sight. The point in which we should naturally expect most uniformity would be the physical condition, since the lesion involves mainly cells which have generally acknowledged functional attributes in the motor mechanism. In this direction as well as with regard to the mental condition and the etiological constellation there is, however, a considerable divergence of the data.

The cases of Turner, Worcester and Wiglesworth and my own were all patients committed to hospitals for insane. Those of Ballet and Hoch died in general hospitals. Those of Ballet were cases of cachectic or alcoholic-tubercular delirium, some of them with more or less evidence of polyneuritic disorders. The case of Folsom and Hoch was one of a protracted apathetic depressive state—a more accurate picture of the general condition cannot be obtained beyond what is contained in the summary. Worcester's patient with "Landry's paralysis" began with a hypochondriacal paranoic condition, and three months later she developed the symptoms of polyneuritis, which terminated fatally in four weeks. Turner's case 4 was one of polyneuritis accompanied by a nervous apprehensive state of one year's duration.

Turner's case 6 was one of "acute delirium" of very rapid course, and not unlike certain cases of peracute general paralysis.

My case 4 was one of alcoholico-senile deterioration of a very uneven course, also my case 8.

The great number of the cases 1, 2, 3, 5, 6 and 7 of mine, and 1, 3, 5, 7, and perhaps also 2 of Turner's, were at the outset depressive psychoses of the period of involution. In Turner's cases 3, 5 and 7, and in my cases 1 and 3, the diagnosis of agitated melancholia was made; Turner's case 1 and probably also 2 are atypical depressions, my case 2 a depressive delirium, case 5 an unusual depressive hallucinatory paranoic condition with a final deterioration resembling general paralysis, and case 6 a case of recurrent hypochondriacal depression with many features of agitation in the second attack.

After having seen the first two cases (1897) I was inclined to think that the condition present was one liable to occur on the ground of senile or pre-senile melancholia (see my foot note in the first communication), and in my differential diagnosis I depended greatly on the existence of this condition, especially when it presented somatic deliria and rapid physical deterioration under neurological and general symptoms not unlike those seen in certain cases of general paralysis with a rapid decay, but without the typical speech defect and less definite neurological alterations. On this ground the diagnosis was made before death in the cases 4 and 5, while in case 6 even the possibility of a lesion was but vaguely entertained, since it was a second attack of a psychosis rather unlike the others. In several cases in which the general picture of the psychosis and the general condition was very suggestive shortly before death, although the motor disorders were quite vague, a diagnosis of probability was ventured upon, but with negative result, so that to-day I should put the chief emphasis on the physical findings and consider the psychosis merely as the expression of a favourable ground. It would lead too far to present here the material of negative cases, although it would prove decidedly instructive. I also omit the cases in which the histological alteration was present only in isolated cells, since these cases require more careful investigation than we have been able to give so far.

Our present knowledge can be summed up as follows. Under conditions which are not as yet clearly understood, usually in the course of certain forms of melancholia or in asthenic conditions, such as alcoholic-senile or alcoholic-phthisical deliria (and perhaps also in the course of true general paralysis?), a peculiar symptom-complex has been found to occur. With a delirious or stuporous mental condition a progressive weakness developes, usually shown first by occasional falls and a stumbling gait. This, of course, fails to be noticed in patients who are already bedridden owing to general reduction of strength or owing to stupor, or asthenic deliria, and it is in no way characteristic since it is also found in certain alcoholic deliria and in states of ordinary weakness. In most cases it is probably not correct to say that there is a paresis in the sense of well localised paralysis. The patients often present a very striking power of resistance, especially in the arms, when they do not display much strength otherwise, on account of the absence of attempts at well-directed spontaneous motility. This condition of weakness, as Turner has pointed out admirably, is frequently, but not always, accompanied by a peculiar motor restlessness, which may vary from a blind general excitement (see Turner's case 6) to a more occasional twitching of the arms, or a subsultus tendinum. It really resembles the physical symptoms of a protracted delirium tremens, rather than the convulsive episodes of general paralysis. These symptoms are frequently very vague and, I suspect, not infrequently overlooked by attendants and physicians or merely described as weakness, or "nervous" or "hysterical" manifestations, such as tremor, peculiar motions, &c. In my sixth case, the attendant reported that the patient had occasional peculiar spasmodic twitchings in his arms, but only when he thought himself watched. The rigidity, too, which occurred at times is hardly characteristic enough. It is very difficult to distinguish it from negativistic resistence of ordinary deliria, and the pain evidenced in Hoch's case on attempts to overcome it is by no means sufficiently characteristic for any special rigidity. The description of the final episode in our case 6, and that of the more lasting condition in our case 7 and in another form in Dr. Hoch's case; and, on the other hand, the total absence of these symptoms of irritation in our case 8, show some of the extremes observed. In a few cases only (Turner, Ballet, and Worcester) polyneuritic symptoms were present. In case 7 there was difficulty in swallowing, and a partial ptosis during the last two days. There were, however, in all cases certain derangements of the *reflex-mechanisms*, either in exaggeration of the tendon-reflexes (most commonly) or absence of the knee-jerk (as in my case 5, without plain polyneuritic symptoms, and Turner's case 4 with polyneuritis). The cutaneous reflexes vary. The plantar reflex is by no means always absent. It is of interest to note that an unequivocal Babinski reflex has not been observed so far. Tests of sensibility are very fallacious, owing to the mental condition. Turner mentions the occurrence of diarrhœa during the later period of the disorder, also observed in several of our patients; our case 7 also showed irregular fluctuations of the temperature without leucocytosis and without Widal-reaction; the others were not continually enough observed, so that we have only a final fever curve associated with the very common terminal broncho-pneumonia.

The vagueness of the symptoms makes it difficult to say anything concerning the *duration*. It seems to be an acute or sub-acute disorder of from two to four or more weeks, usually implanted on a more chronic deterioration. Cases like my third and fifth would almost suggest the possibility of partial remissions.

The anatomical findings. Macroscopically nothing characteristic could be noted in the nervous system. The findings are the same as those met with in cases of the same age and general condition, without the characteristic microscopic alterations. The brain weights, too, vary greatly. The membranes show various degrees of a very moderate thickening of the pia, but no adhesions, and the vessels at times varying degrees of beginning arterio-sclerosis. The first case had a scar of softening in the right caudate nucleus; no other macroscopic lesion of the brain substance was noted. In the same case there was also marked thickening of the skull with hyperostosis of the inside of the frontal bone.

Microscopical findings. The nervous system as a tissue is not markedly altered. Even the thickening of the vessels described by Turner was not present in all cases, and only few small hæmorrhages were found (as in case 1, in the cornu ammonis).

A small-celled infiltration of the blood-vessel sheaths, such as is so extensively present in those cases of general paralysis which show an anatomical lesion, was not seen in any of the cases. The neuroglia was not more abundant than in other brains of the same ages, and certainly not as it is found in general paralysis. We deal with a disorder which does not involve the tissue as a whole, but is mainly a *parenchymatous alteration of certain nerve elements* with remarkably little general reaction of the tissue. The alteration became first obvious on account of the appearance of the cell bodies, and the examination with the

Marchi-reaction showed that many fibres were in a condition of decay of the myelin sheath.

The first description quoted in the introduction mentions my first suspicion that the whole picture of the change in the cell bodies pointed to a lesion of the projection fibres, since it was typical for the "axonal alteration." It became next evident that a lesion of the brain stem was found only in the right caudate nucleus of the first case, that it was accidental and did not account for the bilateral disorder in the Betz-cells. The later cases showed no such coarse lesions, and it became further evident that the typical axonal alteration was found also in cells outside of the cortex. Similar cell changes of the character of axonal reaction had, in the meantime, been published repeatedly concerning the segmental motor neurones in nerve lesions and cases of polyneuritis and in Clarke's columns of cases of tabes and meningitis, and I had drawn attention to the same change in these and several other cell types under less characteristic conditions in my "Critical Review," &c. The examination with the Marchi method yielded positive results in the hands of Turner and Hoch. But when it was applied to our case 6, it showed a marked discrepancy between the number of fibres which have the reaction of degeneration in the pyramidal tracts, and the number of cells with axonal alteration, and this led to a more extensive examination of my cases 7 and 8.

The result shows conclusively that it would be wrong to see in it only a disorder of the cerebral cortex alone. It is a diffuse affection, mainly of elements within the neural tube, but undoubtedly also present in some of the neurones constituting the "peripheral nerves." In several cases (Turner, Worcester, Ballet) the polyneuritic features were indeed quite prominent.

The general histological character of the lesion is the same as what is described in parenchymatous peripheral neuritis or degeneration. From the standpoint of general pathology, we see in this a degenerative process, analogous, but of course not necessarily homologous, with degenerative processess of other cell types, but considerably modified, owing to the peculiar structure of the tissue elements of the nervous system, and especially of the elements most directly under consideration, the nerve-elements or neurones, with their highly differentiated cell bodies and processes and medullary sheaths, &c.

There is, unfortunately, no general term available for this condition. The most common distinctions are those according to localisation. From the "preneuronic" period, we have a very crude way of dividing the nervous system into brain, spinal cord and peripheral nerves. In the peripheral nervous system, we speak of peripheral neuritis, a vague term which includes the reaction to traumatic and infectious and toxic results. Like the type under discussion above, the *parenchymatous peripheral neuritis* does not involve so much the coarse anatomical structure "nerve," as the individual nerve fibres; and when we speak of parenchymatous neuritis of certain sets of nerves, we imply by it a degenerative affection of more or less of the nerve fibres which help constitute the "nerve."

In the central nervous system this condition has attracted attention, mainly in connection with a few chronic diseases of the spinal cord. There it is spoken of as systemic and non-systemic degenerations, involving a more or less great number, either of fibres of a recognised set, or of several sets, or diffusely scattered without any regard for a limitation to such sets. The systemic and polysystemic degenerations in the cord have received their special terminology, usually derived from the clinical picture, and as long as only few "systems" were usually considered, the tracts with degenerations were easily enumerated. For the diffuse degenerations found, for instance, in pernicious anæmia, a more definite description was not attempted, beyond a general localisation of areas in the cord section.

The difficulty makes itself much more felt in those who work on the brain, in which system diseases have been relatively little described outside of the typical secondary degenerations; and those who work with some forms of the neuron concept have additional reason to be dissatisfied with the existing terminology because we cannot stop at the study of bundles of fibres; we must take into consideration that the fibres are parts of cells, and that the correlation of cells and fibres is a prominent postulate of neurological accuracy. The conditions which occurred in our cases illustrate this well. At first we discovered a change in the cell bodies, and now we know, moreover, that there are special sets of corresponding fibres in degeneration. In polyneuritis the degeneration of the myelin sheaths was the first finding; and the cell changes became known later. As we shall presently see the change in the cell is quite characteristic; but its nature, and the nature of its correlation with the alterations of the fibre is hardly explained, not even with the most high-sounding theory.

In a discussion on Landry's paralysis, the writer has already pointed out in 1896, how vague the terms "polyneuritis, myelitis, and encephalitis" are when we deal not with the local disease of the entire tissue of a locality and its remote consequences, but with more general toxic, infectious or asthenic parenchymatous degenerations in the nervous system. The present experience shows that the traditional nomenclature based on coarse anatomical localisation is inadequate for description and didactic purposes. Many cases of peripheral neuritis of a not local origin are probably peripheral only because the other parts of the nervous system are not examined with equally adequate methods as the peripheral nerves. To judge from a rather large material treated with the Marchi method, the extent of decay of medullary sheaths throughout the nervous system is far greater than the old hæmatoxylin methods made one suspect. Unfortunately it is difficult to find a term which would apply indifferently to all degenerated conditions with decay of myelin sheaths of any nerve unit. I propose to adhere to the shortest word, "neuritis." It should not meet many objections, because the concept "inflammation" is more and more dissolved, and one would hardly see anything "inflammatory" behind the syllable "itis." But the terms might seem to refer especially to nerves, and their next analogues, nerve fibres. The very persistence with which the attribute "peripheral" reappears with the word neuritis shows, however, that the use of the term is not as fixed that we should not be allowed to speak of "central neuritis," or in more general conditions of "general neuritis," instead of a long descriptive expression, "diffuse degenerative affection of nerve elements with myelin decay and axonal reaction." It seems probable that the rising generation, with the increased knowledge of the life or nerve elements or neurones acquired since Forel's inspiring paper of 1887, will think more in terms of nerve elements than the generation from which we received our training, and also some of our prejudices. They will get accustomed to think more of the histologically important correlations of parts than of coarse anatomy whenever they are not especially anxious to refer to topographical conditions, such as encephalitis, myelitis, and neuritis of local origin.

There is another feature which we usually think of in connection with the terms neuritis or polyneuritis. We can safely say that no diffuse parenchymatous neuritis is truly general. There are—clinical experience shows this—fairly definite types of distribution more or less characteristic for the origin, so that even in the great diffusions there is usually a certain system. On the other hand, the more accurate methods have shown that system diseases are rarely strictly monosystemic. Tabes does not limit itself absolutely to the intraspinal course of the afferent segmental neurones; amyotrophic lateral sclerosis is not merely a degeneration of the pyramidal fibres, &c. The cases under consideration are classed under the name of *central neuritis* with the understanding that we deal with a parenchymatous degeneration of nerve elements, *mainly* of the *central* nervous system, and that this type is one belonging to the general group of "diffuse neuritis" with a certain degree of systematisation.

Concerning the extent of the central neuritis, I refer to the cases 7 and 8, which show that there are various degrees of the disorder and perhaps also various distributions.

We cannot omit the discussion of a rather difficult point in the problem, namely, the peculiar distribution of the changes within the same nerve elements. This, unfortunately, is a question in which we ought to have at our command a larger material and working force than our State hospital furnishes to-day. Many of the conflicting statements one meets in a digest of the pertinent literature come from the looseness partly of the nomenclature of the writers, partly of the descriptive evidence furnished, so that perhaps the problem is not quite as hopeless as it appears. In my "Critical Review" I pointed out how small our chances are under the best opportunities to really get a sufficiently definite idea of the condition of any entire nerve element, and this makes it especially desirable that results should not pass into the literature without a careful analysis of the validity of the facts as well as the descriptive terms and the definitions adhered to.

As an instance, I quote the very article in which Faure communicates his six cases of cell changes in the cortex in toxic infectious mental disorders quoted above (p. 95). He discusses the question whether the cell alteration is always secondary. Several of his cases had plain polyneuritis, but he used only alcohol methods in the cortex and not Marchi's method, and he remains wisely reserved. He merely adduces one argument against the necessarily secondary character of the cell alterations, which might well be criticised in a few words. Together with Ballet, he sectioned the projection fibres underneath the motor cortex in seven dogs and killed them eight, eleven, eighteen and twenty-one days after the operation. "Comparison of the sections of cortex from the operated and normal side demonstrated plainly the disappearance of the large pyramidal cells." In three cases they also examined with Marchi's method without finding any alteration in the fibres—hence there is a discrepancy with "the complete disappearance of the large pyramids"—or have the fibres too completely disappeared in three weeks? Only one case of the seven (and that one was complicated by infection) did present the axonal alteration (instead of the "disappearance"?). These contradictions invalidate the weight of the communication very seriously and relieve our feeling of standing before another great puzzle. His statements are so flatly at variance with what Bregmann and others have shown and also our own experience concerning the cortex in subcortical lesions, that we are forced to suspect a serious error of observation, and that we regret to see the article quoted without criticism of this glaring contradiction.

A further complication lies in the indiscriminate use of the term "chromatolysis." The expression came into vogue when the stainable portion of the cell body and of the dendrites were newcomers in neurological descriptions, and, perhaps, absorbed undue attention. Since then it has again become evident that in the physiological and pathological life of nerve-cells it is wrong to classify all the changes according to the condition of only one part of the cell. While we cannot claim to be anywhere near a final recognition of the possible changes and their classifications, we are able to-day to speak at least of a few *types* with a possibility of being understood by those who take the trouble to acquaint themselves with well-described distinctions. The most striking types are those of pyrexia (commonly called acute alteration), the axonal reaction present in our cases, Hoch's shrinkage-alteration, the mere rarification of the stainable substance, &c. To classify these as varieties of chromatolysis promotes the existing confusion. The term "central chromatolysis" is utterly misleading, since there are at least two such types, the pigmentary and the axonal reaction, and because it suggests a change *around* the nucleus. By *axonal reaction*, we understand that cell alteration which we can produce experimentally by the section of the axone. It is as it were a focal disorder within the pericaryon; splitting up and dissolution of the stainable bodies usually between the place of origin of the axone and the nucleus (not *around* the nucleus), swelling of this region with involvement of the greater part of the cell-interior and dislocation of the nucleus towards the surface and even beyond the normal contour; persistence of a peripheral ring of lumps, and of the spindles in the dendrites. Decay, or at least fatty alteration of the medullary sheath of the axone is a frequent accompaniment. When this alteration is combined with acute alteration (see p. 108) the stainable bodies of the periphery and dendrites are also dissolved, and the dendrites showed the characteristics of the acute alteration. When we speak of "axonal alteration," we imply that this is the *type* identical with that always observed where section of the axone occurred near enough the cell body to produce any reaction at all; and this *type* we have now found to exist where the nerve fibre was merely diseased as it was already found in polyneuritis, and also under conditions in which disease of all the nerve fibres in question has not been demonstrated, but where at least some fibres show the presence of the general conditions for degeneration of fibres (my case 8 and the instances mentioned in p. 94). It would seem from our cases that the cell body may show the alteration before the fibre is visibly affected, unless we should assume the other possible alternative that many of the *débris* of fibres were already resorbed; and Warrington, Goldschieder and Marinesco claim that the same change may occur if a set of superimposed neurones is affected which is normally the source of stimulation of these cells. It is obvious that the question is still complicated; but this should be a special reason to make the distinction of the type which is already recognised, instead of throwing it together into the hazy "chromatolysis." The axonal reaction to us is an, as yet, unexplained complex of manifestations, which as far as we know now, seems closely related to the constellations under which we recognise also myelin decay. Not all the cell types react equally, and the assumption of a "central neuritis" is probably justified only where entire sets of cells show the reaction without there being any disease of the *tissue* which would account for the occurrence of degeneration after the type of secondary and retrograde degeneration. The frequent occurrence in Clarke's columns and in other cell types requires further studies, and we must admit that we are still far from knowing all the possible histological steps in the process.

The *nosological position* of this condition is as little within the reach of definite decision as the mental disorders which formed the beginning, or as little as other not directly toxic forms of polyneuritis; and this is not the place to rehearse the reasons why we prefer a statement of ignorance to a discourse on auto-intoxications at this stage of "mental pathology." We propose to wait or work for more tangible data, and commit ourselves to the following remarks.

In order to obtain the necessary amount of data concerning frequency of the central parenchymatous nerve decay, we need a larger number of thorough examinations also of the *supraspinal* nervous system in many types and conditions of nervous diseases. I refer here to such possibilities as studies of cortex in polyneuritis or more or less diffuse spinal affections, such as were described by Russell, Batten, and Collier, under the name of subacute combined degeneration of the spinal cord, and to such lines of work as the studies of Zappert on the root and cell changes in the central nervous system of the child, the studies of Kirchgässer on the nervous system in cancer, cachexia, &c., and the publications of Luce. My own experience in the study of the nervous tissues, with modern methods for the observation of the cells as well as the fibres, cautions me against hasty statements. I merely venture to make the remark that our ideas of the normal nervous system, on the one hand, and of what might be called fatal true degenerations on the other, represent extremes, and that our present delicate methods show us stages to which the standards of text-book correlations obtained from less discriminating methods—viz., the law that degeneration implies abolition of function and, probably, irreparability, if found in the central nervous system—should not be applied without much care. There are most probably various degenerative conditions in the central nervous system, not all of clearly-known clinical significance, and some of them may be functionally trivial, and it is quite probable that the Marchi method does not merely show old facts in a more precise garb, but certainly also conditions of less striking importance than the serious degenerations of the more primitive neuro-pathology, which saw degeneration only when it was macroscopically visible. It is especially deplorable that studies of the axones are so difficult in the central nervous system, since the recognition of a disorder of the axone should be furnished in addition to the one of its myelin sheath, before the relation between function and lesion should be discussed. My studies in this direction are hardly mature for publication.

Findings similar to ours have lately been established by Bonhoeffer and by Starlinger. Starlinger describes, in his "Beitrag zur pathologischen Anatomie der progressiven Paralyse" (Monatsschrift of Wernicke and Ziehen, vol. vii., p. 1-13), the presence of fairly-localised degenerations which do not differ much in their general distribution from what we found in our case 7, were it not for their more unilateral occurrence and the difference in the clinical history of the cases and in the condition of the cortex, which Dr. Starlinger kindly demonstrated to me. The latter shows the correctness of his diagnosis with just as much certainty as we should exclude the typical lesion of general paralysis in our own cases. The distinction is probably in the main as follows:—The predominant character of general paralysis is that of a tissue alteration, whereas our findings are strikingly devoid of this feature. According to Starlinger, the distribution of the degenerations in general paralysis is dependent on the extent and localisation of tissue alterations, and it is, therefore, less systemic and less symmetrical. Besides this process, there *may* exist in general paralysis one allied to the type of central neuritis, as case 9 of John Turner's series might suggest. Personally, I have not met a case of true "central neuritis" in clinically and anatomically plainly established general paralysis.

The findings of Bonhoeffer refer to alcoholic delirium (Wernicke's Psychiatrie) and to "Seitenstrangerscheinungen bei acuten Psychosen" (Psychiatrische Abhandlungen, Heft 2, Breslau, 1896). The latter, although observed clinically only, suggest strongly a more frequent occurrence of central alterations than has been commonly surmised, and form a reason why we prefer to be rather conservative as to conclusions. The alcoholic degenerations found by Bonhoeffer are most likely akin to those in our own cases, and we can confirm the observation from a case of plain alcoholic delirium in which, however, the lesion was in no way as extensive as in those of "central neuritis" of non-alcoholic origin described above and in which the cell alteration was far from general.

Turner, in his first description, calls our cases a "form of dementia associated with a definite change in appearance of the pyramidal and giant cells of the brain." Since the above was written he has even ventured to base on these cases his theory "concerning the physical conditions of the nervous system which are necessary for the production of states of melancholia, mania, &c." While we have seen that most of our cases have arisen on a basis characterised by depressive states, we have met at least two cases of alcoholic-senile deterioration in which depression was absent or trifling; and in a sufficient number of cases we have failed to establish all the findings in undoubted instances of profound depression. Neither the expression "dementia" nor the inferences as to melancholia and mania appear to me warranted at this stage of our observation. Hypotheses have a value only if they stimulate to further research, but not if they give premature satisfaction. For this reason I prefer to call the general condition of our patients by the terms "delirium," "stupor," and "coma"—not by the term "dementia," simply because I consider it impossible to establish what deserves the name "dementia" in any of these states, as they prove fatal before an examination for dementia is quite conclusive. We might as well call a more or less profound delirious and stuporous condition of the fever-alterations and of certain intoxications a "dementia." Another question is the relation of these mental states to the preceding melancholia or alcoholic or senile delirium. Here, too, reserve is advisable. As long as we do not know the conditions from the point of view of entities fulfilling the requirement for an admission into true causal chains, we do best to treat them merely as empirical chains of facts.

That the modern views even on "general paralysis of the insane" to-day appear to be in a harbour of dogmatic security, is chiefly due to their plausibility and their value in practice and teaching; yet even they would hardly be able to stand a moderately severe criticism unless we modestly admit their empirical character. The discussion has merely been stopped owing to the difficulty of getting at more accurate facts. For this reason we avoid a long discussion of the question whether our group of cases should be called a type of general paralysis.

Summary.—(1) Eight times in 200 autopsies in which a microscopic inspection of the cortex took place a condition of bilateral changes of the nature of the axonal reaction in practically all the Betz-cells was observed, accompanied by the same changes in other cell types and, where this was looked for, by decay of the medullary sheaths of some of the corresponding sets of fibres, whereas the characteristic disease of the tissue of general paralysis (gliosis, vascular infiltration, &c.) was absent. An extensive examination of large areas of the brain in three cases shows the existence of a partially systemic but widely distributed and strikingly symmetrical parenchymatous alteration of numerous nerve elements, chiefly of the cortico-thalamic connections of the motor area, the auditory radiation, the forceps, the pyramids, the fillet, the restiform body and, to a lesser degree, the posterior columns of the cord, the intersegmental elements, and the segmental efferent motor elements. The change is that of axonal reaction in many of the large cell types and decay of the myelin sheaths. Instead of the long descriptive term, "Partially systemic parenchymatous degeneration principally of the central nervous system," we propose the expression, "central neuritis" in the sense of an equivalent of parenchymatous neuritis, but mainly of central distribution.

(2) This alteration has been found to occur in peculiar forms or end-stages of depressive disorders, near or after the climacteric period, alcoholico-senile and alcoholico-phthisical cachectic states, idiocy, and perhaps also general paralysis (Turner's case). Ordinary infectious and cachectic states do not, however, appear to form an important link in the causes.

(3) Clinically, the symptoms are rather vague: after a

course in which there is no suspicion of organic disorder, there appears, more or less suddenly, difficulty of locomotion, increasing weakness for coordinated movements, at times jactations of the limbs and rigidity and disorders of the reflexes, often together with diarrhœa and occasional febrile fluctuations; the mental condition in this terminal episode is either that of anxious perplexed agitation, delirium, or stupor, similar to a protracted delirium tremens. The nature of the disorder appears to be an equivalent of the general type of parenchymatous nerve-cell alterations, but, in distinction from the well-known infectious and toxic types of "polyneuritis," of pre-eminently central distribution.

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EXPLANATION OF THE FIGURES.

Fig. 1.—Betz-cell from *Case* 1. Axonal reaction. $\frac{1}{12}$ oil immersion, Leitz.

Fig. 1*a*.—Betz-cell from the same case. Vortex conditions of the chromophilic portions. $\frac{1}{12}$ oil immersion.

Fig. 2.—Betz-cell with vortex formation, *Case* 8.

Fig. 2*a*.—Large pyramid of the frontal cortex (*Case* 8), with axonal alterations. $\frac{1}{12}$ oil immersion.

Figs. 3 and 4 (in text).—Right hemisphere of *Case* 7, with the levels of the slices for Marchi specimens: (*a*) through the foot of the frontal lobe;

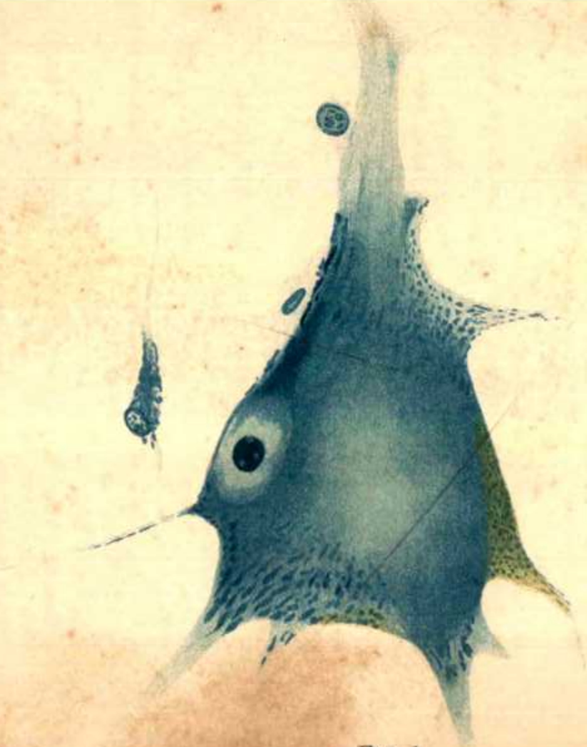

FIG. 1.

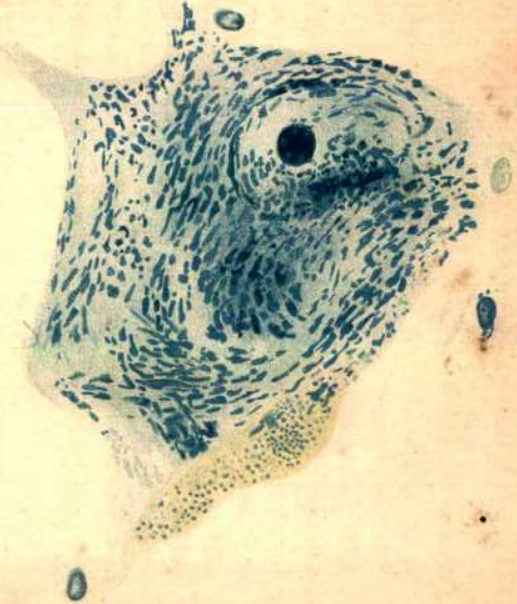

FIG. 1*a*.

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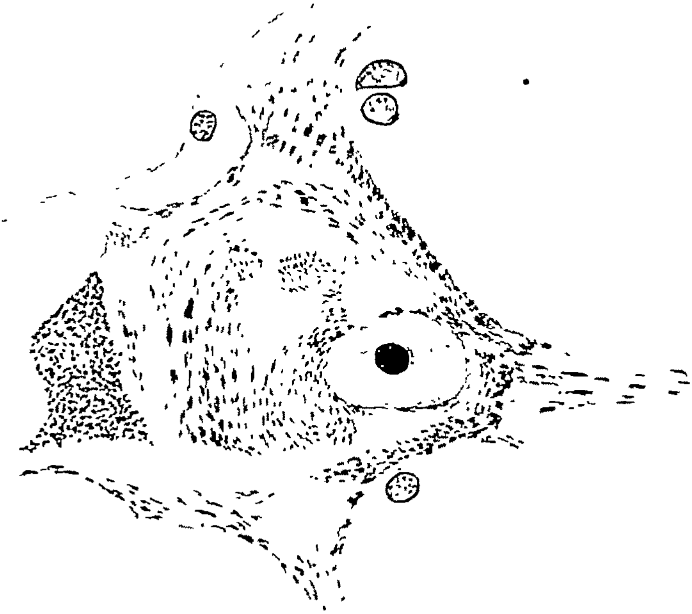

FIG 2

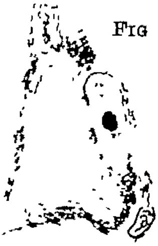

FIG 2a.

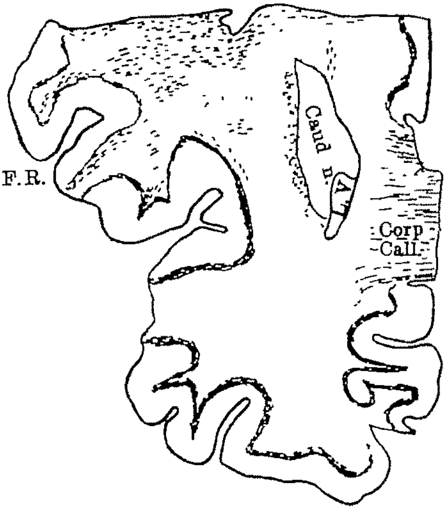

FIG. 5

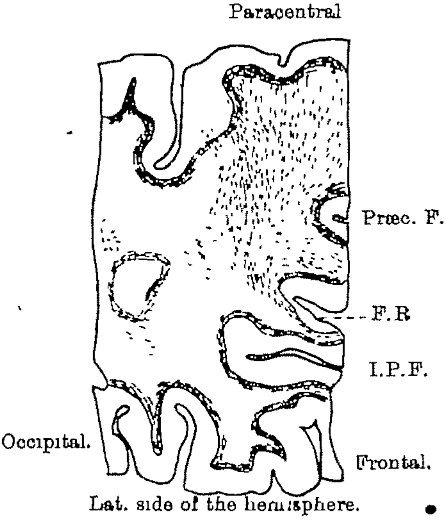

FIG. 6.

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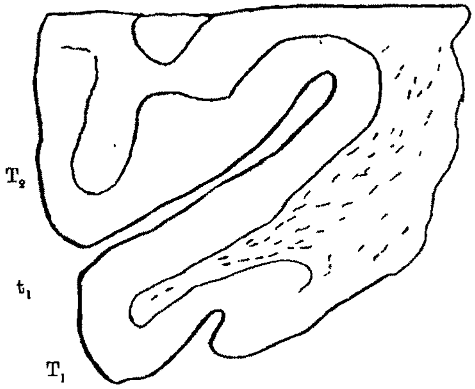

Fig. 7.

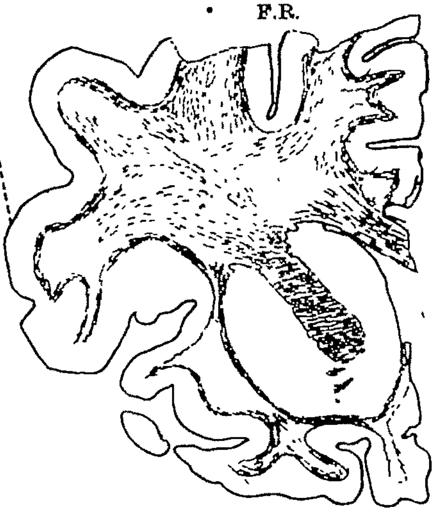

Fig 8.

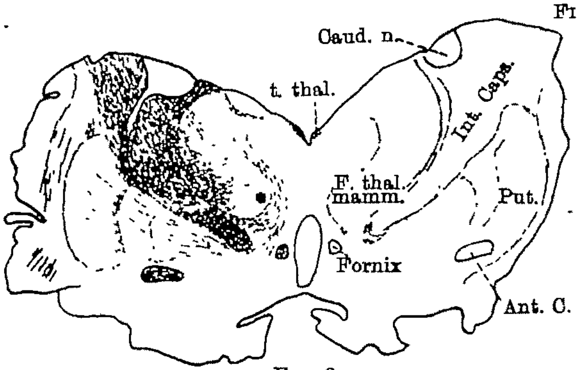

Fig. 9.

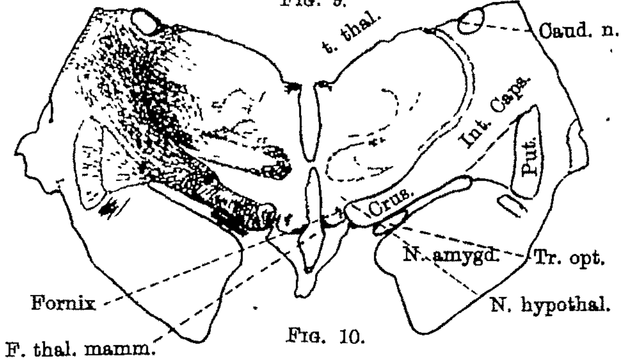

Fig. 10.

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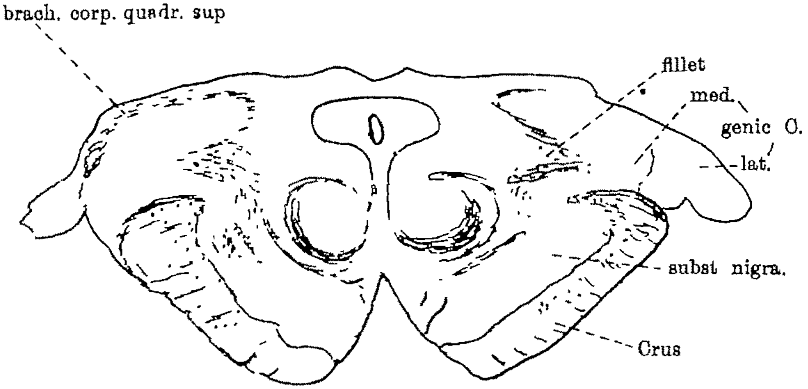

FIG 11.

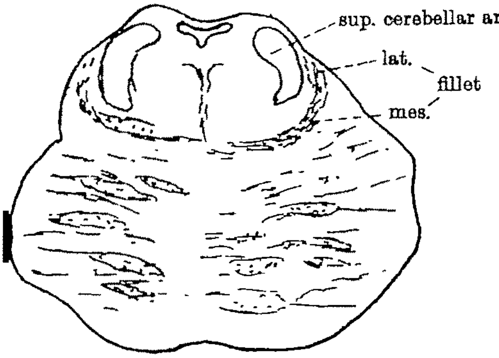

FIG. 12.

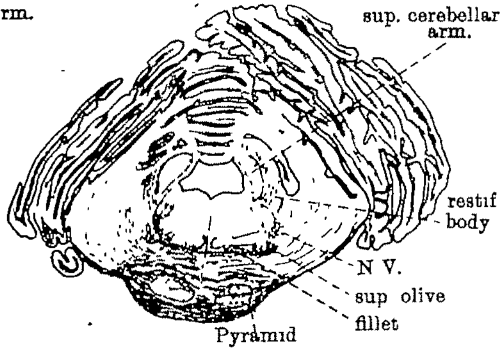

FIG 13

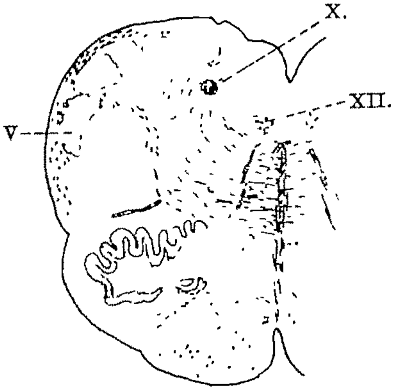

FIG 14.

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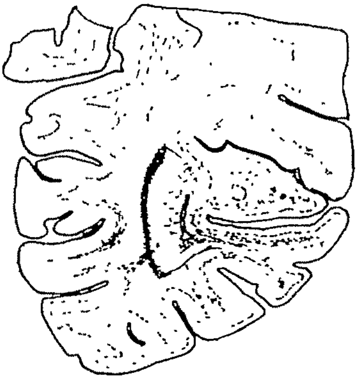

FIG. 15.

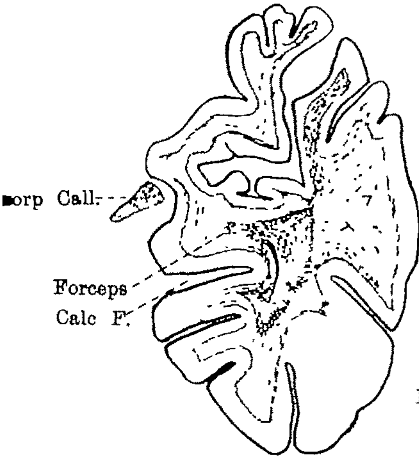

FIG. 16

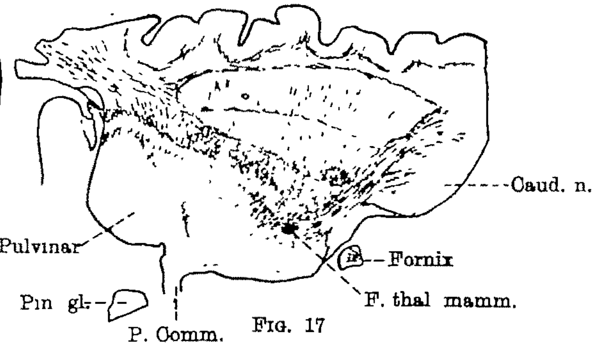

FIG. 17

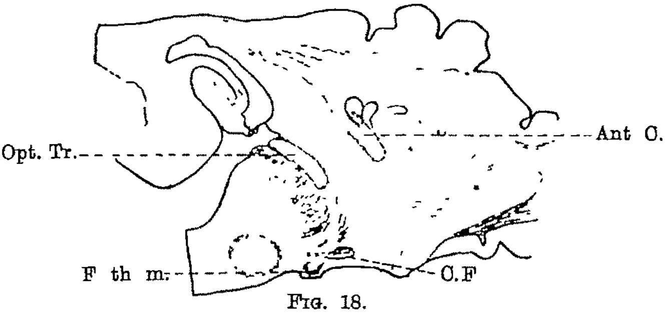

FIG. 18.

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(*b*) underneath the paracentral lobe; (*c*) through the temporal lobe; (*d*) frontal section through the motor area and in front of the anterior commissure.

FIG. 5.—Section from fig. 3*a*. Degenerating fibres limited to the motor area and its part of the centrum ovale, and of the corpus callosum.

FIG. 6.—Section from fig. 3*b*. Degenerating fibres limited to the projection system of the motor area. Only few fibres from the parietal lobe.

FIG. 7.—Section from fig. 3*c*. Degenerating fibres limited to T_1.

FIG. 8.—Section from fig. 3*d*. Degenerating fibres from the motor cortex to the dorsal part of the internal capsule and the corpus callosum.

FIG. 9.—Section from the anterior part of the thalamus (only the left side drawn; the whole being symmetrical). Degeneration of fibres of the internal capsule and its radiation into the lateral thalamic nucleus only.

FIG. 10.—Section from a posterior level. Division of the degenerating fibres into a radiation of the ventro-lateral nucleus of the thalamus and pyramidal fibres.

FIG. 11—Mid-brain, with degeneration of pyramidal fibres only in the crus, few fibres in the fillet, and few fibres in the arm of the superior corpus quadrigeminum.

FIG. 12.—Section through the posterior corpus quadrigeminum. Degenerating fibres in the pyramid and fillet.

FIG. 13.—Section through the trigeminal segment. Degenerating fibres in the pyramid, the corpus restiforme, and few in the fillet.

FIG. 14.—Section through the pneumogastric segment. Degenerating fibres in the pyramid, the corpus restiforme, and few in the fillet.

FIG. 15.—Section from the left occipital lobe. Degeneration practically limited to the forceps and its radiation.

FIG. 16.—The same on the right side, with evidence of degeneration in the splenium of the corpus callosum.

FIG. 17.—Horizontal section through the left thalamus of *Case* 8. Degeneration limited to the internal capsule and the radiation of the anterior half of the lateral thalamic nucleus; scanty degeneration in the radiation of Gratiolet.

FIG. 18.—Section parallel with the last one. Very scanty degeneration, limited to the pyramidal tract.

SECTION OF THE POSTERIOR SPINAL ROOTS FOR THE RELIEF OF PAIN IN A CASE OF NEURITIS OF THE BRACHIAL PLEXUS; CESSATION OF PAIN IN THE AFFECTED AREA; LATER DEVELOPMENT OF BROWN-SEQUARD'S PARALYSIS AS A RESULT OF LAMINECTOMY; UNUSUAL DISTRIBUTION OF ROOT ANÆSTHESIA; LATER PARTIAL RETURN OF SENSIBILITY.[1]

BY MORTON PRINCE, M.D.,

Physician for Nervous Diseases to the Boston City Hospital (U.S.A.).

THE report of this case is published because it shows:—

(*a*) The areas of anæsthesia following section of the fifth, sixth, and seventh cervical spinal roots.

(*b*) The relief of pain as a result of the operation.

(*c*) Development of Brown-Sequard's paralysis following laminectomy.

(*d*) Partial return of sensation in previously absolutely anæsthetic areas.

Jeremiah F., as a result of being struck by a railroad train, received a Colles' fracture of the left wrist, and developed a complete and absolute paralysis of nearly the whole left arm. The paralysis was typical in its distribution, in its course, in the electrical reactions (R.D.), atrophy, &c., of a neuritis of the brachial plexus. The paralysis has continued ever since, and the arm has been useless from the shoulder down. The absolute paralysis involved all the muscles of the shoulder, upper arm and forearm, but the intrinsic muscles of the hand and the pronators of the forearm were only partially paralysed. There was diminution of sensation over limited areas, which will later be described. The most intensely affected muscles were the upper arm group, namely, the supra and infra spinati, deltoid triceps and bicipital group, for the absolute paralysis of these muscles continued at

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[1] Read at the meeting of American Neurological Association, May, 1900.

the end of a year and a half after the injury, with marked atrophy and loss of the electrical reactions, while at the end of a little over a year some power had returned in the forearm muscles, for at the end of this time Jerry could flex and extend both the wrist and fingers, though with very slight strength. There was also slight power of pronation. From this it will be seen that all three cords of the brachial plexus were involved. The most diseased fibres were from the fifth, sixth, and seventh cervical segments, while those coming from the eighth cervical and first dorsal, supplying the intrinsic hand muscles and the long flexors and extensors of the fingers, were partially implicated. The heft of the injury was therefore to the fibres that make up the supra scapularis, musculo-cutaneous, circumflex nerves and triceps portion of the musculo-spiral nerve, and therefore to that portion of the plexus which is made up of fibres from the fifth, sixth, and seventh cervical. At any rate, the fifth, sixth, and seventh cervical fibres were most severely injured, while of these three the fifth and sixth suffered the most.

From the time the splint was taken off Jerry suffered most intensely from pain which was located chiefly in the thumb and forefinger. For this he was referred to the Clinic for Nervous Diseases, Boston City Hospital, where he was under treatment for a long time. For the relief of this pain, which was exceedingly intense and justified almost any operation that promised relief, three different operations were undertaken at different times, but none of them seem to have been based on sound neurological principles. About two months after the accident the radial was stretched in the forearm, under the theory that the pain might have been due to injury of the nerve at the wrist by the fracture; but the operation failed to give relief. About seven months later he was operated on in another hospital. The exact nature of the operation could not be ascertained, but an incision was made along the course of the nerves in the upper arm, it is said with the idea of removing a "callus." Later still, about fifteen months after the accident, he again entered the surgical ward of the City Hospital, and a second incision was made in the upper arm, in the region of the brachial plexus, and what was thought to be a piece of diseased nerve removed, but which turned out to be a piece of fibrous tissue. Shortly after this (October, 1899), he was again referred to me by Dr. J. C. Munro, in whose service he was at the time. At this time the paralysis was as has already been described, and the arm was perfectly useless for practical purposes. Whatever strength had been regained in the hand could not be utilised because of the intense pain in the thumb and forefinger already spoken of. A rough touch to these digits caused the most excruciating and unbearable pain. The hand and arm had to be handled with the greatest care, and was carefully nursed and kept supported by the patient. He expressed himself as willing to undergo any operation for

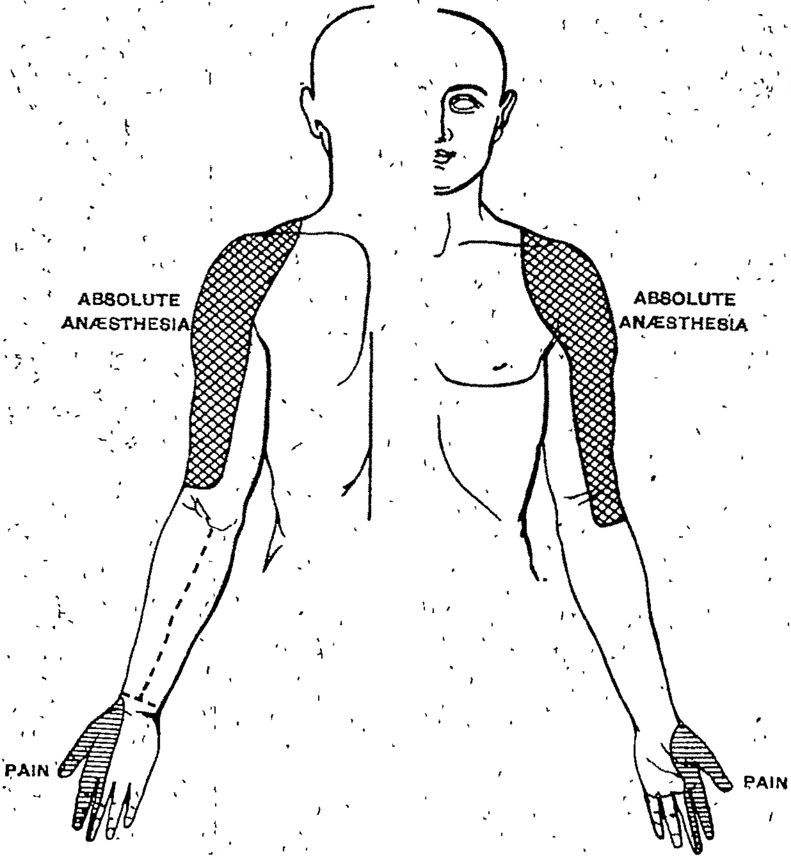


DIAGRAM I.

Showing location of pain in the hand prior to operation; also area of absolute anæsthesia at same period.

relief. The distribution of the pain may be seen by reference to diagram I. It was most intense and disabling in the thumb and forefinger and corresponding half of middle finger, back and front, but there was another line of pain of much less intensity and importance extending like a half-bracelet around the back of the wrist, and from the middle point of this bracelet up the middle of the forearm to the olecranon. It will thus be seen that the severe pain in the thumb and forefinger did not correspond in distribution to the area of any nerve supply, but did correspond to the usually accepted distribution of the sixth cervical root or segment.

I accordingly advised as a rational procedure, and the only one likely to give relief, section of the posterior roots of the fifth, sixth, and seventh cervical nerves, the fifth and seventh being included in case some of these fibres overlapped in the affected area, and therefore might be the source of the trouble. Laminectomy was accordingly done by Dr. J. C. Munro. The operation was a very severe and bloody one—one of the most difficult I ever witnessed, in consequence of the fact that the patient was a thickset man with a "bull neck." From the moment of coming out of the ether Jerry was completely free from pain in all the previously affected areas, the wound healed perfectly by first intention, and the operation was a success for the purposes for which it was undertaken.

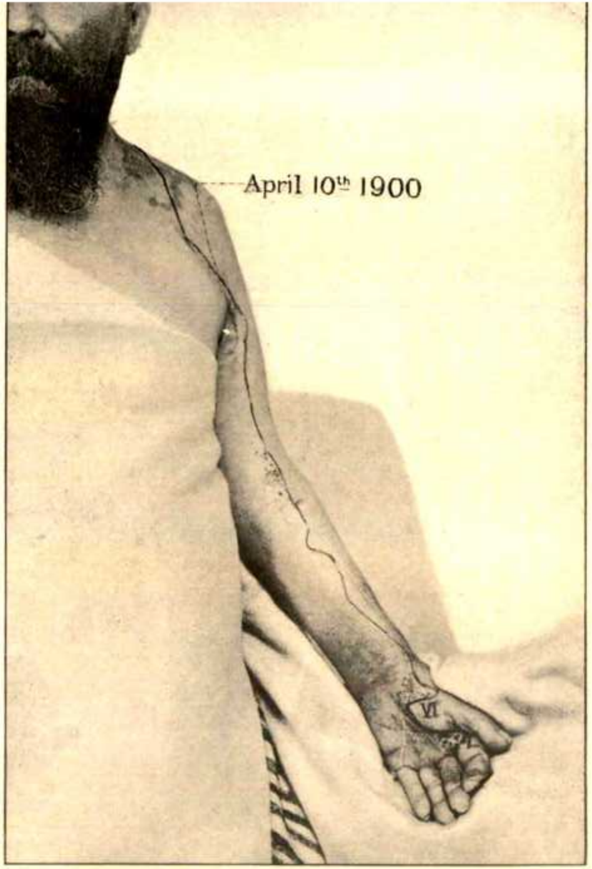
Jerry's troubles, however, did not end here. After he began to get about it was noticed that he exhibited a limp and weakness of the left leg, and examination showed that there was a moderate Brown-Sequard paralysis. The left leg was decidedly spastic and paretic. There was a marked increase of the deep reflexes of this leg including an ankle clonus, and he could not raise his weight on the leg, while there was a moderate diminution of sensibility over the whole right leg. This anæsthesia was limited above by the groin; on the left sensation was normal, as were muscular power and the reflexes on the right. During the operation the danger of a hæmorrhagic clot being partially formed within the spinal canal occurred to me, as indeed it had at previous operations of this character, although I had never known it to occur. I may say here, merely to emphasise this point, that I called attention to this danger at the time of the operation. Now this possibility was suggested anew by the occurrence of the paralysis. Nevertheless, it is equally likely that the paralysis was due to pressure from granulation tissue or some form of callus. As the exact time of onset of the Brown-Sequard paralysis is not known, it is not easy to decide between the two possibilities. That the paralysis is due to pressure of some sort and the sequence of the laminectomy is scarcely questionable. It is noteworthy that the pressure on the cord is on the side (left) on which the roots were cut.

But still more trouble awaited Jerry. There gradually developed severe pain on each side of the neck behind, so that turning the head to either side caused great pain, of which he complained considerably. Thus it happened that at the time of his discharge from the hospital, about three months after the operation, he still suffered from severe pain, though in a different region from that originally affected. The cause of the pain in the neck was undoubtedly a neuritis, due to some nerve being injured either by the operation or by the healing thereof. Although the arm itself was free from pain, it was perfectly useless, and the patient expressed a strong desire to have it amputated to get it out of the way. He has been promised a consideration of this request if, at the end of another year, he has regained no practical use of it.

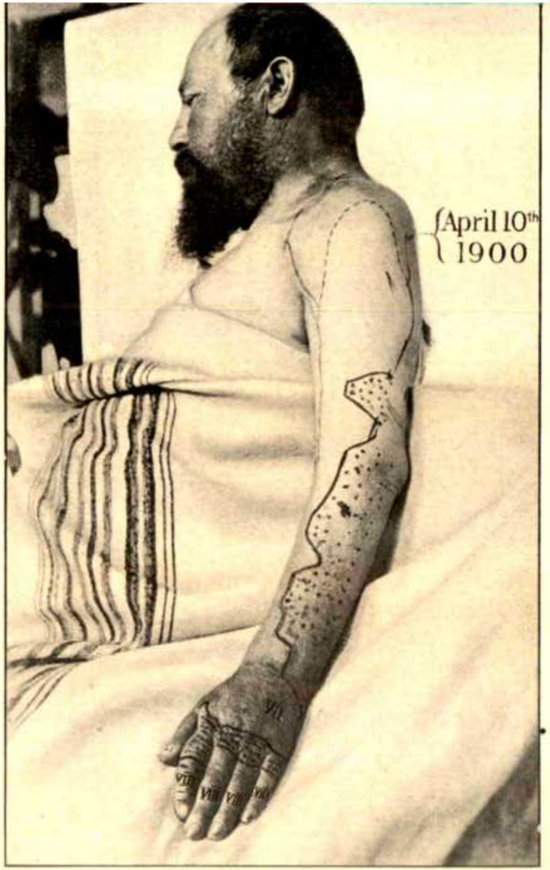
A recent examination of Jerry (April 10, 1900), about five months after the operation, shows a great improvement in his condition, and that the operation has been a success for the purpose for which it was undertaken. He is completely free from pain in his hand and arm, and the pain from the neuritis in the neck has also ceased. He can turn his head freely without discomfort. He is now free from pain so far as his original trouble is concerned, but he does have pains about the shoulder, which apparently are due to the weight of the paralysed arm dragging upon the parts. By supporting the arm this pain is relieved. But the arm remains completely paralysed from the shoulder down, and is absolutely useless. The paralysis of the hand has rather increased, so that he can little more than move the fingers. The electrical reactions have almost completely vanished. On the other hand the Brown-Sequard paralysis has improved very much, so that he walks without perceptible limp, although the left leg is still weaker than its fellow; he can go up and down stairs only with care, and he cannot raise his weight by his left foot or leg. There is still moderate anæsthesia in the right. The limiting line of the anæsthesia is at the height of the groin as before. An uncomfortable symptom, and one of some interest, is a sensation of intense coldness in the right foot. He still desires to have the arm amputated.

ANÆSTHESIA.

The distribution of the anæsthesia following the section of the spinal roots was very interesting. It differed materially from that which would be expected from the diagrams of Starr, Kocher, and Wichmann. It was studied with great care.



PHOTOGRAPH A.



PHOTOGRAPH B.

In the forearm and hand the area (radial aspect) included within the heavy lines is that in which absolute anæsthesia developed after section. Above it joined the previously anæsthetic area in the shoulder. The dotted area represents diminished sensibility on the back of the forearm. This existed previous to operation. The broken line over the shoulder indicates the degree of return of sensibility April 10.

Several charts of the anæsthetic areas were taken at different periods previous to the final operation. They all were in substantial agreement excepting that the chart taken just before the section of the posterior roots showed a new area of absolute anæsthesia (diagram I.) covering the

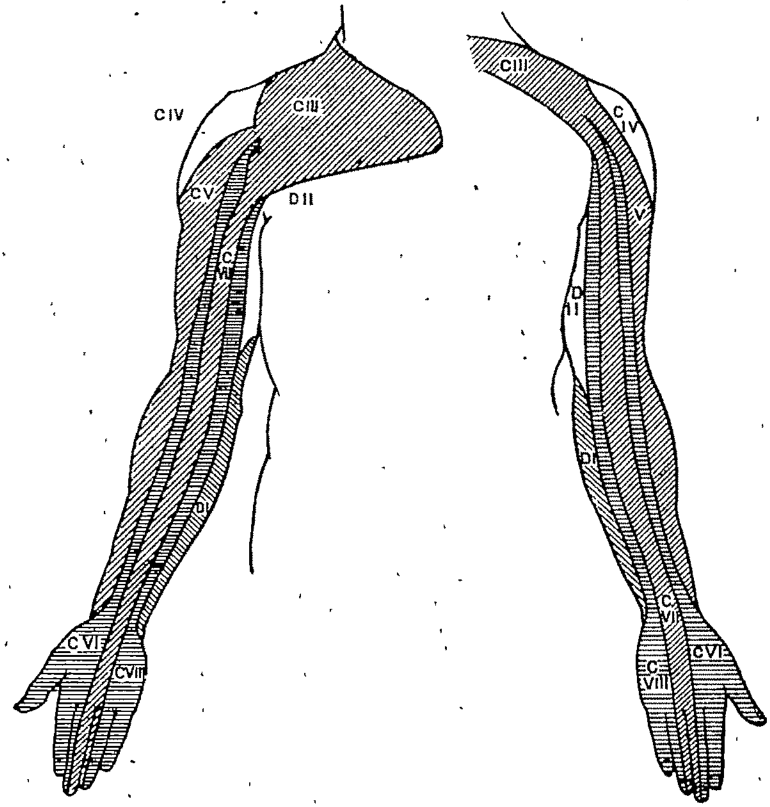


DIAGRAM II.

After Starr. To show cutaneous root-areas over front and back of arm and hand.

shoulder and extending above the clavicle about half-way up the neck, and below over the corresponding upper two-thirds of the arm. Whether this was due to one of the operations, or to an extension of the neuritis, is not clear. Its presence was important only so far as it made it impossible to determine the upper limits of the distribution of the fifth root, as the anæsthesia from the latter merged into this already existing anæsthetic area. Besides this, there was in the forearm and lower third of the upper arm, previous to section of the roots, and as a result of the neuritis, an area of impaired sensibility. This impairment was limited to the radial aspect, and was continued down over the thumb and forefinger. It included both pain and touch, but not in coextensive limits. As analgesia it extended over the whole of the thumb, but only over the back of the forefinger and back of the hand. As tactile anæsthesia, it spread over the thumb and palmar aspect of the fore and middle fingers, and a portion of the back of the hand.[1]

But, with the exception of that over the shoulder, neither the analgesia or the anæsthesia was anywhere absolute. For the most part, the anæsthesia consisted of numbness, that is to say, the patient *felt a light touch*, but the feeling was described as "numb." The analgesia was deeper than the tactile anæsthesia, but as tested by a pin prick was not absolute. It was most intense in the *whole* thumb, back of hand and back of the forefinger, that is, those areas of the hand which (excepting the forefinger) became absolutely anæsthetic after section of the roots. The difference between this slight or moderate impairment of sensibility and the absolute loss of appreciation of touch and pain which in parts of the same areas followed the section of the spinal roots, allows us to recognise the cutaneous distribution of the latter.

According to the usual description (diagram II.) of the root areas, the absolute and complete anæsthesia, after section of the roots, besides including the radial side of the arm (V.), should have covered the thumb, forefinger and adjoining half of the mid-finger (VI.), with only impaired sensibility, perhaps, over the remainder of the mid-finger and adjoining half of ring finger;[2] this latter because the area of the seventh would have received additional fibres from the adjoining eighth cervical or first dorsal. This is not, however, exactly what happened. *The absolute loss of sensation for touch and pain was limited to the whole thumb with its ball, back of the hand and a strip along the radial aspect of the forearm and arm* (see photographs). Over the other fingers the impairment was not perceptibly increased over the slight numbness that was present before operation; excepting that over the backs of the proximal phalanges there was a zone (dotted in photograph) of diminished sensibility for pain and touch, which was not noticed before the operation, and over the whole back of the forefinger the analgesia was increased, but tactile sense was unchanged, over the remainder of the fingers sensibility was not changed from what it was previous to the section of the roots. It is particularly noteworthy that the *palmar surface of fore and middle fingers*, a region generally given to the sixth root, was *not* anæsthetic.

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[1] It is interesting to note that this total area corresponds fairly closely with the areas of the fifth, sixth, and seventh roots as given by Starr, Kocher, and Wichmann. The diagrams of these writers, however, differ considerably in the limits of the areas allotted to each of these three roots separately.

[2] Kocher includes even the ring finger in the sixth root area.

The following conclusions as to the root supply, then, seem to be justifiable in this case:—

(1) The absolutely anæsthetic strip along the radial edge of the forearm corresponded with that ascribed by both Starr and Kocher to the fifth root and must have been due in part or wholly to the section of this root.[1]

(2) The sixth root in the hand could not have supplied more than the thumb (front and back) with its ball, though it probably aided in giving sensory fibres to the *back* of the forefinger (increased loss of pain sense). This is a much more limited area than that usually given to the sixth cervical,[2] as will be seen by comparison with diagram II.

(3) The seventh cervical *probably* gave a more extensive supply to the back of the hand than usual, but not to the fingers. It may have contributed to the partially anæsthetic areas over the first phalanges of the fingers. The further distribution of the seventh, either to the palmar or the

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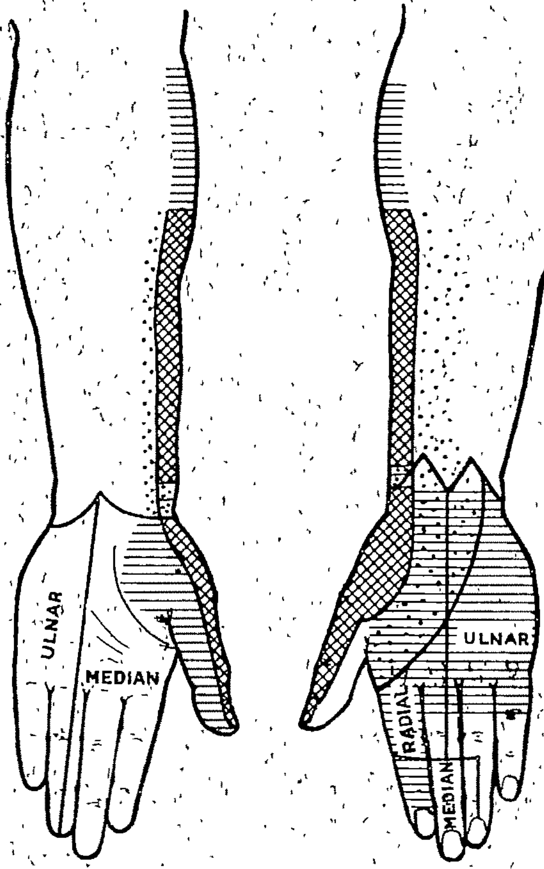
[1] The section of the sixth root may have contributed to this area as given by Wichmann.

[2] As the seventh was cut, the preservation of sensation over the fore and middle fingers could not have been due to overlapping of this root.

dorsal surfaces, was not demonstrable. The area of impaired sensibility along the back of the forearm (dotted in the photograph) could not have been due to section of the seventh cervical, as it was present before the operation.

(4) The remainder of the backs of all four fingers and



DIAGRAMS III. AND IV.

Taken seven months after operation and showing the relation of the anæsthetic areas to the distribution of the ulnar, median and radial nerves; also restoration of sensibility. Cross-shading = loss of pain and tactile sense; transverse lines = analgesia; dotted areas = diminished tactile sense (in forearm the limits were not determined at this date). Tactile sensibility had returned over ball and palmar surface of thumb and partially or wholly over a large part of back of hand. The analgesia is substantially that immediately following sections of the fifth, sixth and seventh cervical roots.

nearly the whole of the palm and palmar surfaces of the fingers must have received a supply from the eighth cervical or first dorsal, or both, which would thus have a wider distribution than that usually assigned to them.

The probable explanation of these apparent variations in the root areas is to be found in a corresponding variation in the termination of the ulnar and median nerves. The ulnar nerve is supposed to supply ordinarily the inner third of the palmar surface of the hand (diagram III.), but variations occur. This nerve derives its fibres from the eighth cervical and first dorsal roots, and if it had an unusually wide distribution in the hand, these roots would supply areas ordinarily given to the sixth and seventh cervical through the median; or possibly the eighth cervical root fibres contained in the median might be more widely distributed than usual, and some be continued into the branch of the median that supplies the area in question. The variation shown on the back of the hand is still more interesting. Here the fingers showed no increased anæsthesia, excepting near the proximal knuckles, and even here it was not absolute. Now the median (diagram IV.) supplies a quadrangle covering the distal half of the backs of the first three fingers approximately. It is presumable, therefore, that in this case the fibres which the median derives from the eighth cervical must have been distributed to this quadrangle, and not those from the sixth and seventh, which were cut, as would be the case according to the diagram of Starr and others; and further, that the median had a wider distribution over the whole of the backs of the fingers. A reference to diagram III. will make this clear.

Very remarkable was a later return of sensibility in previously anæsthetic areas. On April 10, about five months after the operation, it was noted that above the shoulder the anæsthetic area had retreated from the neck to the shoulder-joint, and corresponded with the circumflex nerve area (dotted line in the photograph); in the arm sensation had crept in to some extent on the lateral borders. In the hand, likewise, tactile sensibility was returning. Two months later still (June 22), a quite extensive return of the tactile sense had occurred. This Jerry himself had noticed by testing himself.

He feels the lightest touch over the ball and palmar aspect of the thumb, though here there is still analgesia. Only over the dorsum of the thumb is there absolute loss of both pain and tactile sense. Over the dorsum of the hand he feels a very light touch to the ulnar side of a curved line shown in diagram III., but between this line and the area of absolute loss of sensation he feels a firm touch only. The analgesia remains as before. In the forearm there has also been considerable return of the tactile sense. As the roots were freely cut, it is difficult to suppose they could have re-united; the return of sensibility must therefore be due to overlapping nerve supply from adjoining root areas, which have become functionally more exciteable. It is not easy to explain the return of tactile sense, without improvement in the sensibility for pain. As to the Brown-Sequard paralysis, he complains bitterly of a burning feeling in the *right* thigh and coldness of the right foot; if cold water is dropped on any part of the leg he says it feels like a "hot iron." There is no sensory irritation on the left. The left knee-jerk is still lively, and, although there is no ankle clonus, there is a violent Babinski accompanied by a similar dorsal flexion of the foot and trepidation of foot and leg. In right foot there is a comparatively slight Babinski. The paresis of *left* leg is about the same as when last noted.

A CASE OF BILATERAL PORENCEPHALY.

BY J. WIGLESWORTH, M.D., F.R.C.P.LOND.

THE subject of this case was a female idiot, aged 24, who died in Rainhill Asylum on May 16, 1899, after a residence of nine years. Her parents were natives of Queenstown and each of them was 38 years of age at the time of marriage; they were steady sober people. A first cousin—the daughter of a maternal uncle—died of melancholia in Cork Asylum, but this was the only case of mental or nervous disease in the family so far as could be ascertained, though there had been several cases of phthisis. There were three children from the marriage, two girls and a boy, patient being the third child; the other two were all right mentally, but the eldest girl was not strong physically. All the confinements were easy ones, and there had been no miscarriages. Patient is described as having been always a backward child, and was late in walking and speaking. When a year old she had a bad fall down stairs, with, however (the father said), no visible ill effects. At the age of 3 she had a fit for the first time and continued to have these about every three months or so until she was five years old, when they ceased; the fits were severe and appear to have been of an ordinary epileptic character. During this period what intelligence she had gradually failed, and she does not appear to have improved with the cessation of the fits, for her condition is said to have remained very stationary from that time. A few months previous to admission she had what are described as attacks of "stupor." She began to menstruate when 13 years of age.

When admitted she was found to be a small undersized girl, weighing only 6 stone 12 pounds. She had considerable paresis of the right arm and leg, but could walk, although with a shuffling gait, dragging her right foot after her, and she had a certain amount of power in the right arm, though she seldom used it. She had double strabismus. Mentally she was a decided idiot. She was usually very dull and lethargic, sitting about silent, listless and unoccupied. She never spoke spontaneously in an intelligent fashion, but when spoken to she repeated over and over again some of the words addressed to her in a parrot-like manner without attempting to reply; similarly she would sometimes shout out snatches of songs which she heard other patients singing, and she would often laugh and chatter to herself in a meaningless way. She was never vicious, impulsive or violent. She was at all times wet and dirty in her habits and required a good deal of attention, having to be washed and dressed; she fed herself, however, using both hands for this purpose. At times she had attacks of restless excitement, when she was noisy, destructive and degraded in her habits, plastering herself with her fæces. In spite, however, of her fatuous appearance she understood many simple things that were told her, and when at her best could be got to do a little simple work, such as carrying things about. She eventually died of phthisis

AUTOPSY (*an examination of the head only could be obtained*).

Circumference of head 21½ inches.

Skull, slightly asymmetrical, the temporal and occipital fossæ being apparently larger on the right side than the left.

Encephalon (after draining off all fluid) weighed 852 grammes.

Left hemisphere (fig. 1).—Almost the whole of the outer aspect of the posterior two-fifths, or rather more, of this hemisphere, is converted into a large thin-walled cyst communicating freely with the left ventricle, and in fact forming with this one large cavity; the outer wall of this cyst is for the most part exceedingly thin, consisting merely of thickened pia-arachnoid and ependyma (probably) but in

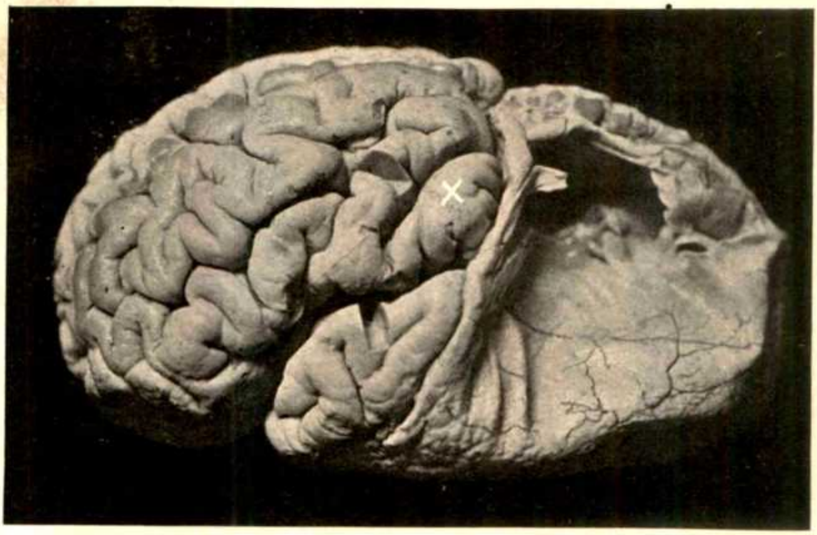


FIG. 1.
Left hemisphere. The × is placed on the lower part of the ascending parietal convolution.

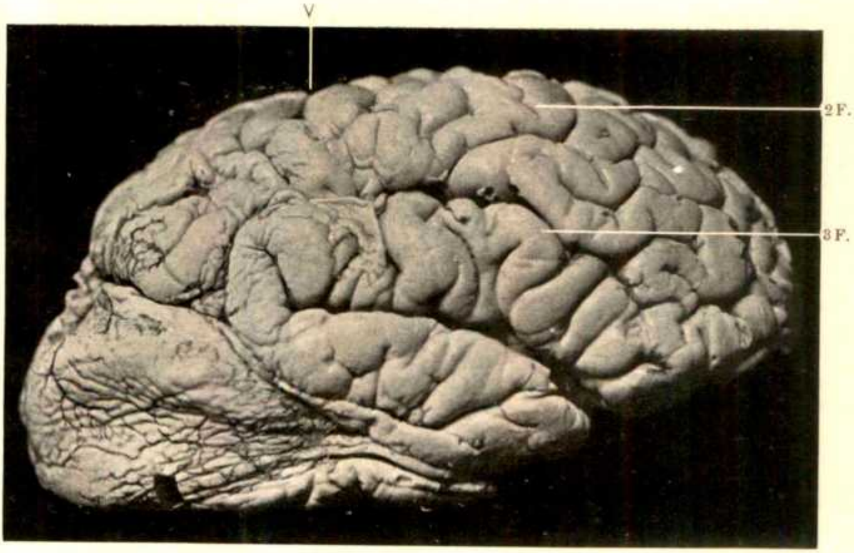


FIG. 2.
Right hemisphere. The arrow indicates the upper end of the fissure of Rolando.
2 F. = 2nd frontal convolution. 3 F. = 3rd frontal.

places, especially as the base of the cyst is approached, it is formed in part also of thinned out cerebral tissue. The cyst is bounded in front at the point where it abuts on the healthy brain tissue by a well-defined line, which is for the most part nearly vertical, cutting the fissure of Rolando across at about its middle, but curving slightly forward at its lower end where it runs into the temporo-sphenoidal lobe. Over the whole of this area the convolutions have entirely disappeared with the exception of a band of puckered and atrophied convolutions at the upper margin of the hemisphere, which appears to represent the remains of the paracentral and superior parietal lobules. The convolutions destroyed therefore comprise the upper third of the ascending frontal, the upper half of the ascending parietal, nearly the whole of the superior parietal lobule, and the external occipital convolutions, a small fragment of the posterior convolutions of the insula, the whole of the supra-marginal and angular gyri, and the posterior half or rather more of the first and second temporo-sphenoidal convolutions, whilst the anterior half of this latter gyrus, together with the whole of the third temporo-sphenoidal, are greatly thinned; on the basal aspect the convolutions entering into the fusiform and lingual lobules (forming the floor of the cyst) are also greatly atrophied, though still showing some remains of their original division; the hippocampal and uncinate gyri are, however, intact, as also is the anterior half (about) of the first temporo-sphenoidal gyrus. The whole of the convolutions on the outer aspect of the hemisphere, in front of the vertical line above mentioned, including the convolutions of the orbital surface, the anterior half of the first temporo-sphenoidal, and almost the whole of the insula, are intact and well developed; whilst on the inner aspect of the hemisphere the convolutions for about the anterior two-thirds are also well developed; but those comprising the quadrate lobule, and more especially those of the cuneus, are distinctly thinned and have lost their normal arrangement, running from above downwards in more or less of a radiating fashion, those of the cuneus indeed being so thinned out as to be undecipherable.

Right hemisphere (fig. 2).—This shows a somewhat similar lesion to that in the left hemisphere, but the destruction of brain tissue is here much less, and the cyst which is formed by a hollowing out of the occipital and temporo-sphenoidal lobes is correspondingly smaller, although forming with the dilated posterior part of the lateral ventricle a cavity of considerable size. The wall of the cyst, too, is not so thin as is that in the left hemisphere, consisting of a layer of thinned-out brain tissue, together with pia-arachnoid, and being nowhere formed by membrane only; at the tip of the occipital lobe, however, where the wall is thinnest, it is barely 1 mm. in thickness. The stress of the lesion has fallen on the convolutions of the occipital lobe, the posterior part of the parietal lobe (more especially the angular gyrus) and part of the posterior portion of the temporo-sphenoidal lobe, the part of this lobe which has suffered most being the area comprised in the second temporo-sphenoidal convolution, which is extensively destroyed, forming a tongue-shaped band of atrophied brain tissue running forwards between the first and third temporo-sphenoidal convolutions, the former of which is intact and the latter fairly so, especially in the anterior half, but it thins off considerably towards its posterior extremity where it forms part of the lower and outer wall of the cyst. Over the area just named which forms the outer wall of the cyst, the convolutions are quite undecipherable, the brain tissue which remains consisting merely of a thin smoothed-out layer varying from 1 to 3 mm. in thickness. Interiorly the convolutions of the cuneus are also much puckered and atrophied, and those comprising the fusiform and lingual lobules, forming the floor of the cyst, are also atrophied, although to a less extent; the hippocampal and uncinate gyri appear normal as in the opposite hemisphere. In front of the area just described as forming the main seat of the lesion, the convolutions of the parietal lobe are found to be much puckered and contracted, and they have lost their normal arrangement, those which have suffered least being the upper two-thirds of the ascending parietal and the superior parietal lobule. The first temporo-sphenoidal gyrus is intact and the supra-marginal gyrus, though small, is also distinct. Anteriorly to the fissure of Rolando the convolutions of the outer aspect of the hemisphere are all plump and well-formed. The convolutions of the frontal lobe however, though well developed, are abnormal in their distribution since they run back in parallel lines right to the fissure of Rolando, and the ascending frontal convolution being thus broken up by the sulci which join this fissure, cannot be distinguished as a distinct and separate convolution throughout its length; there is, moreover, no distinct præcentral sulcus. The orbital convolutions are normal, as are also those of the insula. On the inner aspect of the hemisphere, the convolutions are well developed as far back as the parieto-occipital fissure, and present a fairly normal arrangement except that the convolutions of the quadrate lobule run in parallel lines towards the posterior part of the corpus callosum.

Pons presented no marked asymmetry to the naked eye, but on microscopical examination the fibres of the pyramidal tract on the left side are found to be distinctly smaller than those on the right.

Medulla shows pronounced asymmetry, the pyramid on the left side above the decussation being about half the size of the right one.

Cerebellum, normal; no marked asymmetry.

Microscopical Examination of Cerebral Cortex (made by Dr. A. W. Campbell).

Sections were taken from about the middle of the following convolutions, the method of Nissl being employed for displaying the nerve-cells, and the nerve-fibres being dyed with hæmatoxylin, after fixing with osmic acid or iron alum.

Frontal second. Right.—Depth of cortex, 2·941 mm.; the reduction of depth is uniform, there being no striking wasting of any particular layer. The nerve-cells appear numerically deficient, but not to a pronounced degree, and the layers are arranged in a normal manner. In cells of sufficient size to allow of an examination of the arrangement of the chromophilic particles (larger pyramidal cells), it is found that these elements, though present, stain indistinctly. There is a perceptible reduction in the nerve-fibres in all parts; the tangential band consists only of about half a dozen fibres: the supra- and intraradiary horizontal fibres (Baillarger's lines) are poorly represented, and the vertical striæ of Meynert are thin, and also widely set apart.

Ascending frontal. Left.—Cortex at summit, 3·383 mm. in depth. The nerve-cells are normally arranged, but again deficient in number. The large pyramidal cells show a normal arrangement of chromophilic particles. The tangential band of nerve-fibres is poorly represented, but otherwise the cortex is well supplied with fibres.

Paracentral lobule. Right.—Cortex at summit, 2·55 mm. in depth. The nerve-cells and nerve-fibres are both somewhat deficient. The large multipolar nerve-cells appear to be normal.

Temporal first. Left.—Depth of cortex at summit of convolution, 3·40 mm. Nerve-cells again deficient in number. There is a good tangential band of fibres, but the horizontally directed supra- and intraradiary fibres are not present in normal abundance, and Meynert's striæ are thin.

Temporal third. Right.—These sections are taken from an area where the cortex and white matter together measure only 2·397 mm. in depth; of this, 1·154 mm. represent cortex, the rest white substance and ependyma. In spite of the extreme thinning of the cortex, the nerve-cells, although diminished in number, are yet present in considerable abundance, and their disposition in layers is not altogether destroyed; but they are all of diminutive size and mostly of pear shape, with an apical process only. The body of the cell is mainly occupied by nucleus, and chromophilic particles are not recognisable in the cell-body. In the position normally occupied by the tangential band there is a dense feltwork of nerve-fibres interlacing in all directions. Elsewhere there are few horizontally directed fibres, but there are many vertical ones; these, however, run singly, and are not collected into bundles, as they are in the normal radiations of Meynert.

REMARKS.

Cases in which porencephalic defects occur on both sides of the brain, though less numerous than unilateral ones, are nevertheless not altogether uncommon; thus Audry found the lesion to have existed on both sides in one-third of his collected cases[1] (thirty-two out of ninety-six). That the loss of cerebral substance in this case was due, not to a developmental defect, but to a definite lesion, appears to be clear, though it is less easy to feel sure as to the exact nature of the lesion. There can be little doubt, however, that it occurred either during intrauterine life or in early infancy. There seems to have been nothing in the actual process of birth to have favoured a traumatic origin, and though there is a history of a somewhat severe fall down stairs when the patient was a year old, we have the definite statement of the patient's father that this fall produced no visible ill effects, and therefore it is difficult to associate the lesions with an injury at that period, even though these occurred in parts of the brain which would perhaps least readily give rise to symptoms in an infant of that age. On the whole it seems probable that the cerebral defects in this case owe their origin to obstructive vascular lesions occurring in intrauterine life. Though the area of brain destroyed does not follow with complete exactness the usual lines of arterial distribution, there is, of course, considerable variation in the vascular distribution in different brains, and it may be said generally that those portions of the brain supplied by the anterior cerebral arteries and the anterior branches of the middle cerebral have escaped, whilst the stress of the loss has fallen on the parts to which the posterior cerebrals and the posterior branches of the middle cerebral are distributed. The lesion in the right hemisphere differs from that in the left hemisphere chiefly in the facts that it occupies a smaller area and that the destruction of the brain tissue involved has not progressed to the same extent as in the left. In no part of the area involved in this (right) hemisphere does the brain tissue seem to have disappeared entirely, although in places it is so very greatly reduced in thickness; and this fact favours the idea that the process has, in part at any rate, been a gradual atrophic change due to a slowly-progressive or partial obliteration of vessels, rather than to a single sudden lesion, which would have totally destroyed the brain tissue over a limited area. In the left hemisphere, however, the cerebral substance has gone altogether over a considerable area, and the completeness of disappearance very probably stands in relation to the greater activity of the absorptive process in these early cases, as compared with what obtains in later life. The fact that there was an appreciable deficiency in the number of nerve-cells and fibres, even in those parts of the cortex which appeared normal to the naked eye, is an interesting point, but one which might have been anticipated, as with so considerable an area of the brain practically non-existent, it could not be supposed that the portions which remained could develop the same wealth of connections as in a normal brain.

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[1] "Les Porencéphalies," *Revue de Médicine*, June and July, 1888.

FRIEDREICH'S PARALYSIS.

BY T. DUNCAN GREENLEES, M.B.EDIN., F.R.S.E.
AND
G. CARRINGTON PURVIS, M.D.EDIN., B.SC.

FRIEDREICH'S ataxy is one of those forms of chronic spinal paralyses, standing midway between disseminated sclerosis and locomotor ataxy, and which is considered a comparatively rare disease.

The cases here described are, so far as we can ascertain, the first published in South Africa, and indicate, as has been previously pointed out, that the European inhabitants of this country are rather prone to degenerate nerve lesions.

The following cases occurred under our care at the Chronic Sick Hospital, Grahamstown, in a boy and girl—brother and sister. The girl died last May, and we were fortunate in securing a post-mortem examination. The boy still lives, and a careful clinical examination has been made of him. We are thus able to present a double picture—of the clinical symptoms, as found in the boy, and a microscopic description of the lesions, as found in the case of the girl. The symptoms, in both cases, were so nearly identical that we think the following description can be easily made to apply to both, and that, whereas we regret being unable to give the clinical picture history of the girl, we are at the same time in a similar position as regards the pathological details which may be found in the case of the boy.

CLINICAL DESCRIPTION.

Family history.—Careful enquiries were made to ascertain the family history of our patients, from their mother, with the special object of ascertaining whether there existed a hereditary tendency to nerve troubles. The parents are not blood-related to each other, as so often occurs amongst the Dutch of this country; they are reported both to be healthy, and the father is, or was, fighting for the Transvaal. They had nine children, all of whom were in good health, except our patients, and one child who died from "convulsions" in infancy. The father is reported to be steady and temperate in his habits, is a poor farmer, and lives a healthy out-door life. The mother is in good health and of average intelligence. There are no known cases of paralysis in the family.

Onset of illness.—When Charlie was 9, and Nellie 12 years of age, they both had scarlatina, and while convalescing they got wet one day. It was noticed, shortly afterwards, that both children were weak in their ankles, and gradually the feet began to contract so as to make walking well nigh impossible. The disease progressed very slowly at first, but the parents noticed that both children were steadily getting worse. The contraction of the feet increased, and paralysis gradually affected both legs and arms so that the movements of the limbs became more and more limited in their range, and they became very helpless accordingly. They were admitted from Kimberley on March 11, 1896, being then 13 and 16 years of age respectively.

At that time the girl was very helpless indeed; she was unable to assist herself in anything, and had to be dressed and undressed and fed by the nurse. She sat on a chair all day with her head resting backwards on a pillow; gradually the general feebleness increased; she had several fainting attacks, and during the last few months of her life she had difficulty in swallowing and the respiratory functions became interfered with without pulmonary disease at all. The dysphagia was so distressing that, towards the end of her life, she was quite unable to take nourishment by the mouth.

That the disease, in her case, was one which ultimately implicated the higher nerve centres was evident from the symptoms which, in the end, proved fatal. The difficulty in

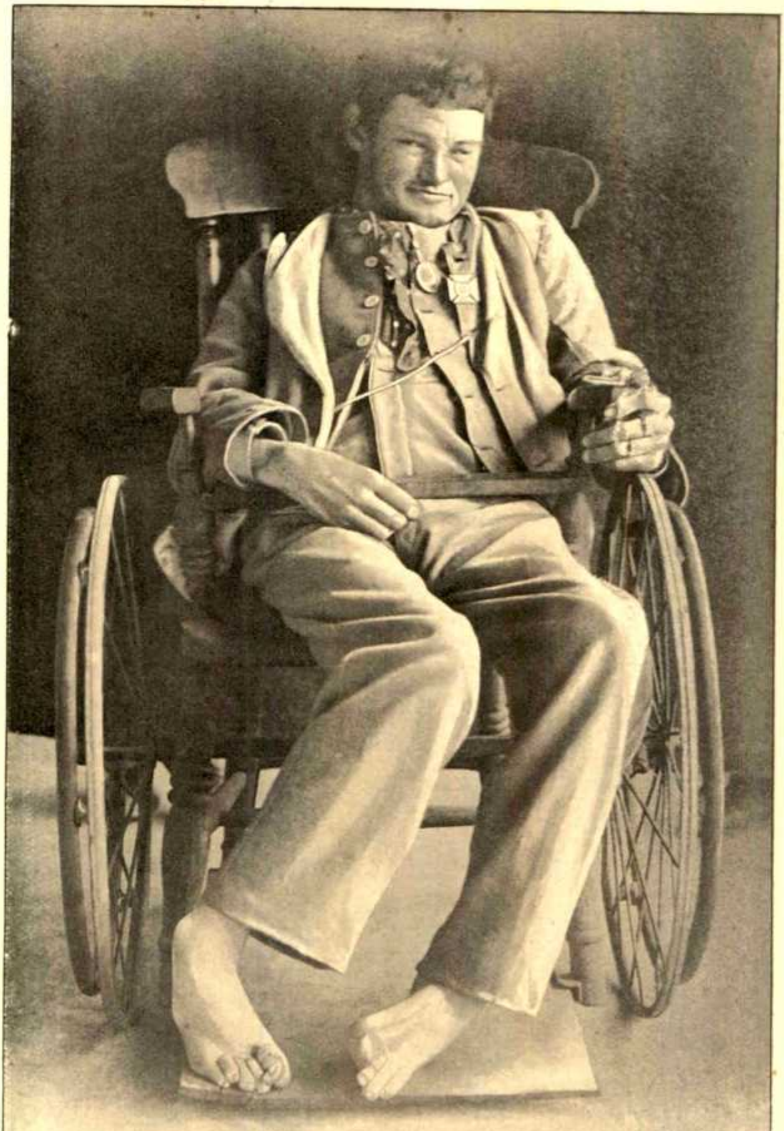

FIG. 1

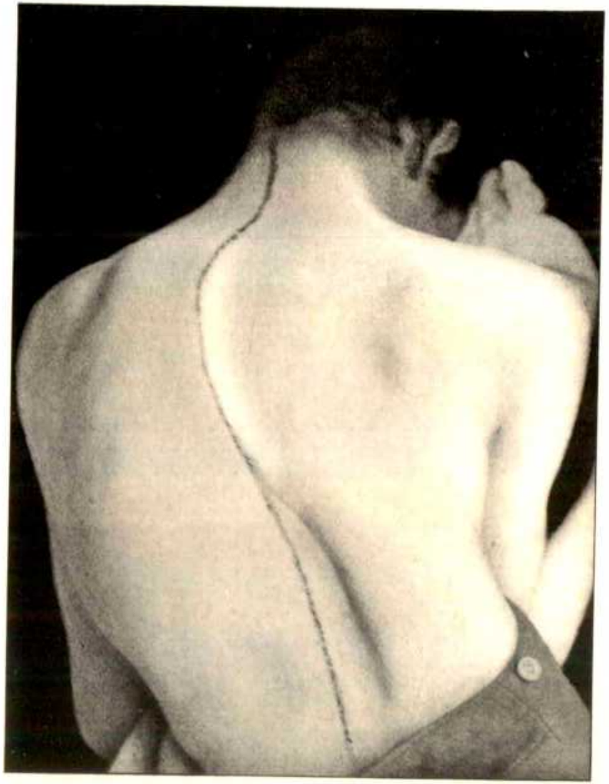

FIG. 2.

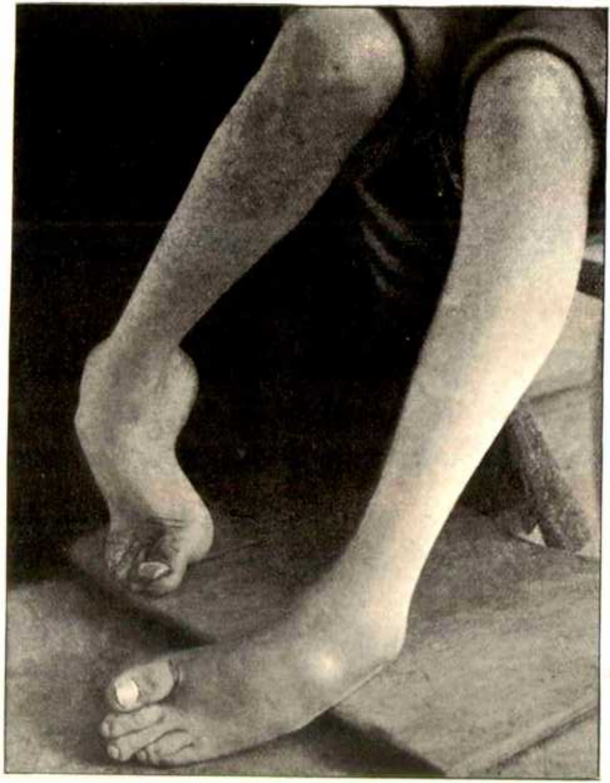

FIG. 3.

breathing, the fainting attacks, and the dysphagia, all indicated some lesion affecting the organic centre in the medulla, and this idea was borne out by the microscopic examination of this portion of the nervous system.

Clinical examination of Charlie.

Charlie is a boy now (October, 1900) aged 17 years. The expression of his face is that of happiness and simplicity combined—almost amounting to imbecility. His face is broad and flat, he always wears a childish smile, and he never loses his temper, nor looks cross. He is quite unable to articulate, but he knows what is said to him. His conduct generally is good, he is very patient, and a favourite with the other imates of the hospital, who are always ready to assist the helpless boy. As is general with cases of slight mental defect, he is somewhat vain, and likes to wear cheap medals, brass chains, and rings on his fingers.

He is utterly helpless, is unable to walk, and, when moved or carried, his head falls limply from side to side as if the neck muscles were unable to support it. While there is a general wasting of the muscles there is no marked impoverishment of nutrition, and his appetite is very good.

His legs hang helplessly down, and when moved about he grips tight hold of the arms of the chair lest he should fall. When at rest both feet are slightly turned inwards, and there is well marked talipes equinus of both feet. This condition is more evident in the right foot, as also is a turning inwards of the foot so that the sole faces almost upwards—approaching to the condition of talipes varus. The great toes of both feet are hyper-extended, a condition likewise most marked in the right side (figs. 1 and 3).

The Spinal Column.—An examination of the back reveals marked lateral curvature, the convexity occurring in the upper dorsal region, and points towards the left side. The apex of the curvature approaches very closely to the posterior angle of the scapula (fig. 2).

Electric Reactions.

With galvanism:

(1) *Flexors of forearms.*

20 cells: Ma = 3.
KCC + ; ACC + ; AOC 0; KOC 0.
Note.—KCC slightly stronger than ACC.

27 cells: Ma = 4·3.
KCC + ; ACC + ; AOC 0; KOC 0.

27 cells: Ma = 4·75.
KCC + strongly marked; ACC + strongly marked;
AOC + very slightly marked; KOC 0.

32 cells; Ma = 10·5.
KCC + strongly marked; ACC + strongly marked;
AOC + slightly marked; KOC + very slightly marked.

(2) *Leg muscles* (posterior and outer aspects).

20 cells: Ma = 2.
KCC + ; ACC 0; AOC 0; KOC 0.

25 cells: Ma = 3.
KCC + ; ACC + ; AOC 0; KOC 0.

30 cells: Ma = 6·5.
KCC + ; ACC + ; AOC 0; KOC 0;

35 cells: Ma = 10.
KCC + ; ACC + ; AOC 0; KOC 0.

It will be seen that the electrical examination—so far as it goes—presents reactions which are compatible, or nearly so, with health.

On applying the *Faradic current* it was noted that the muscles of both arms and legs were slow in responding to the interrupted current; as regards the legs a fairly strong current was required to make the muscles contract.

Reflexes.

(1) *Superficial.*—The plantar, cremasteric and abdominal superficial reflexes were tested and were only slightly

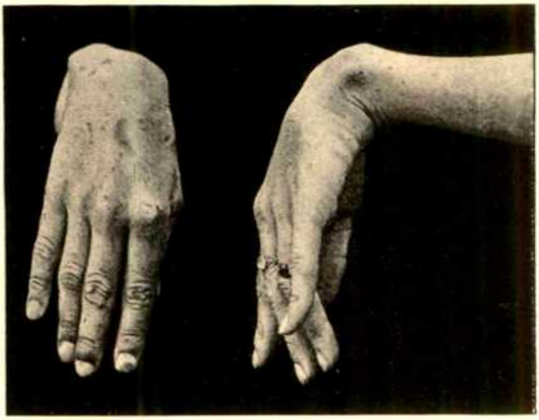

FIG. 4.

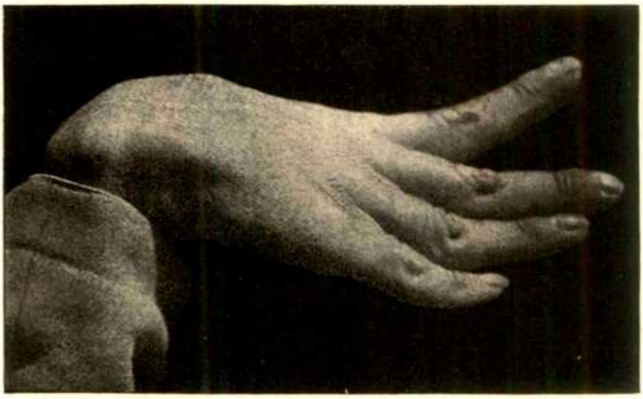

FIG. 5.

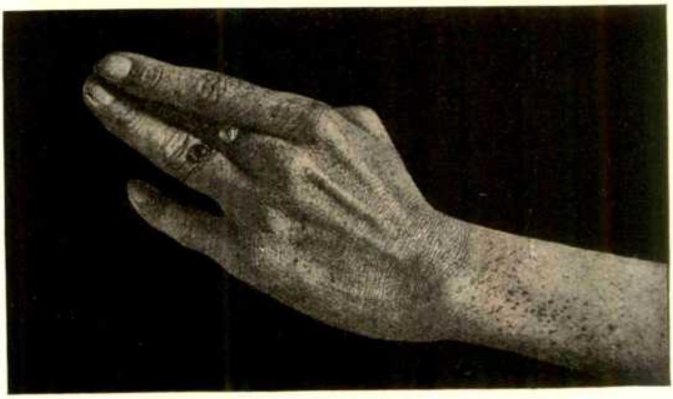

FIG. 6.

evident, while the epigastric, gluteal and interscapular reflexes could not be elicited.

(2) *Deep.*—Both the patellar tendon reflex and the ankle clonus were absent.

(3) *Organic.*—All the organic reflexes were normal except the act of micturition which was somewhat delayed.

The hot sponge test.—The patient felt it hot in the lumbar region as well as in the dorsal region, but only when first applied. The test failed on repetition.

Hot tube test.—Was not felt when applied to either the legs or the arms.

Pain sensation.—The patient felt a needle-prick in various parts of the body, but the needle had to be firmly pressed into the skin.

Muscular sense.—He was unable to differentiate between weights. When asked to touch the tip of his nose rapidly with his finger, he failed to do so, but when allowed to take his own time the act was slowly performed, his hand swaying from side to side while his finger approached his nose.

When told to hold out his hands he presented distinct drop wrist, but when he is asked to straighten them out the fingers are extended backwards to an extraordinary extent. This hyper-extension of the fingers is most marked in the right hand. There is wasting and probably some paresis of the interossei muscles of both hands (figs. 4, 5 and 6).

On being asked to put out his tongue he does so in the middle line; the tongue is large, flabby, and there are general tremulous movements of the organ.

His pupils react readily to light and accommodation, and there is slight nystagmus of both eyes.[1]

The other senses seem perfectly normal, and he complains of no objective symptoms such as pains in the head or sounds in his ears.

PATHOLOGY OF CASE OF FRIEDREICH'S DISEASE.

The following remarks are founded upon the *post-mortem* examination of Nellie B., the sister of the above patient, who died at the Chronic Sick Hospital on May 10, 1900, after a residence of four years, and in whom the disease had existed for nine years.

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[1] Nystagmus was a marked symptom in patient's sister.

Autopsy (twelve hours after death).—Body of a female European girl, much emaciated and muscles wasted. Rigor mortis present. Double talipes equino-varus marked. Small bedsore over sacrum. There was some wasting of the cerebral convolutions, with an excess of fluid in the cranial cavity. The veins of the pia were congested.

The heart and lungs were healthy except for some congestion of the lower lobe of the right lung, and the bronchial tubes contained frothy material.

With the exception of a small cyst in the centre of the left kidney, the abdominal organs presented nothing calling for special note.

A piece of the left frontal convolution, the crus cerebri, medulla oblongata, a portion of the great sciatic nerve, and portions of the cervical, dorsal, and lumbar regions of the spinal cord were placed in Müller's fluid, and forwarded to the Bacteriological Institute, Grahamstown. These were hardened and stained according to Weigert's method, and microscopic sections made.

Examination of the Spinal Cord.

An examination of sections from different portions of the spinal cord revealed the fact, which was even quite apparent to a naked-eye inspection, that the cord was much smaller than it should be. It was quite infantile in size, and it looked even smaller than a healthy cat's cord, with which it was compared.

Microscopical Examination.

Sections from various levels in the cervical, dorsal, and lumbar regions were examined microscopically, and revealed the following pathological changes :—

Cervical region at the level of the sixth cervical vertebra :—

(1) Sclerosis of the middle portion of the posterior internal tract (Goll's tract).

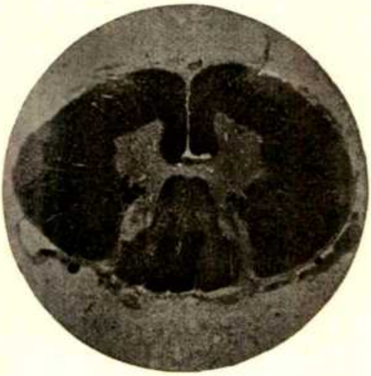
FIG. 7.

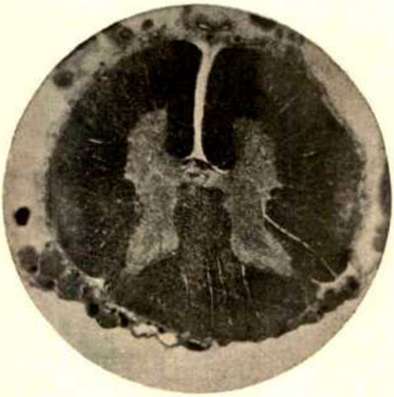
FIG. 8.

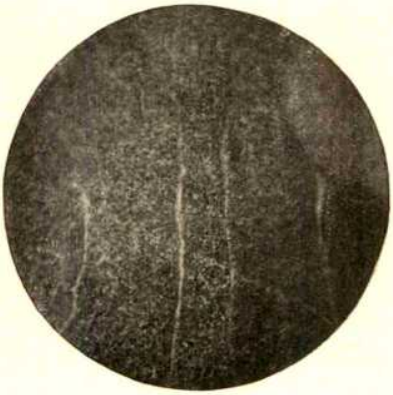
FIG. 9.

(2) Sclerosis of the posterior external tract (Burdach's tract) external to the comma tract.

(3) Sclerosis of the ascending antero-lateral tract (Gowers' tract). This occurs as a small patch of sclerosis just where the anterior nerve-roots leave the cord.

(4) Sclerosis of the direct cerebellar (lateral) tract. This occurs on both sides of the cord to a marked extent, nearly all the nerve-fibres being replaced by fibrous tissue.

(5) In Clarke's column the cells were few in number, markedly atrophied, took the stain badly, and the intercellular tissue was much broken down.

(6) Lissauer's tract, with the exception of a small portion adjoining the direct cerebellar tract, was healthy, as also was the crossed pyramidal tract.

Dorsal region at the level of the second dorsal vertebra:—

The lesions described as occurring in the cervical region were evident, but not to so marked an extent. The sclerosis of the posterior portion of the direct cerebellar (lateral) tract was, however, even more marked, and occurred as a distinct patch, under a low power, of lighter hue than the surrounding healthy nerve-fibres, and this condition was equally marked on both sides of the section (fig. 7).

Clarke's column was affected to a similar extent as existed in the cervical region; its cells and fibres were both affected extensively.

Lumbar region at the level of the third lumbar vertebra:—

In this portion of the cord the sclerosis was very evident, and quite localised to a patch on either side of the posterior median fissure, embracing Bruce's and the comma tract, but leaving the free edge of the fissure healthy. A small patch of sclerosis likewise existed on either side of the posterior fissure more centrally situated and close to the commissure.

The degenerative changes in the lateral and anterior columns, although present, were not so evident as they were in the cervical region of the cord (figs. 8 and 9).

When a higher power was employed it was noted that, in some parts of the diseased portions of the cord, the nerve sheath appeared simply empty of its axis-cylinders. In other portions the axis was either shrivelled or else swollen.

As a result of the former condition, a honeycomb-like arrangement existed, consisting of empty nerve sheaths closely packed together, but where the disease appeared more advanced even the nerve sheaths became obliterated, and replaced by fibrous tissue, so broken down in places as to leave large, almost empty spaces.

The nerve-cells, for the most part, stand out prominently in all the sections; indeed, this prominence is unusually marked where it does exist. In the dorsal region, while the cells of one anterior horn were sharply defined and of a deep orange colour, those in the other horn were hardly evident at all and of a faint primrose colour, and were apparently atrophied, their dendrites and axons being shrivelled. Whereas in the former horn we counted, in one section, six large well-defined cells, the corresponding horn on the other side only showed two pale yellow cells.

The horn in which these faintly-stained cells were found was perceptibly smaller than the opposite anterior horn.

A further examination of these cells revealed several interesting points in cellular pathology. The faintly outlined cells appeared to be swollen, their nuclei indistinct and in some cells pushed out so as to form a prominence in the contour of the cell, and the nucleolus alone appeared sharply defined as a shining point. The contents of the cells were granular and there was an entire absence of Nissl-bodies or any attempt at an intracellular structure.

The cells, on the other hand, which presented a prominent appearance in the various sections, differed entirely in every respect from the others. They were sharply defined, of a darker orange colour, and in contour looked like orange-coloured black berries. They were more rounded than exists in health and their sharp edges had disappeared. In few examples were either axon or dendrite apparent. Nissl-bodies were absent, and chromatolysis in most of the cells was complete. Their contour was so peculiar and unusual that in no drawings illustrative of the pathological changes occurring in nerve-cells, given by Ford Robertson in BRAIN, 1899, and Ewing in *Archives of Neurology and Psycho-Pathology*, Vol. I., No. 3, have we discovered anything resembling the changes herein met with.

We can hardly consider this cellular change—mulberry-like alteration in its contour—to be due merely to "yellow globular degeneration," which, according to Colucci, is due to a morbid metabolism in the cell contents. The change is a change in the general shape of the cell whatever its cause may be, and is not altogether, at all events, the same pigmentary degeneration as that which is quite consistent with health.

Lugaro maintains, and perhaps rightly, that the displacement of the nucleus, as if it were pushed out from the body of the cell, bulging its contour in one place, is due to destruction of the chromatic reticulum which normally holds the nucleus in its central position, and Schäfer holds that the degeneration of nerve-cells in this, and similar spinal diseases, is due to degeneration of the posterior roots and consequent loss of the stimuli normally received therefrom. In fact, that loss of function first produces atrophy of the cell and ultimately degeneration of its contents.

The blood-vessels, as seen in the various sections, were full of blood, and their coats did not appear to be thickened to any extent.

The *Canalis centralis* was somewhat larger than normal, its lining epithelium appeared distinctly and was apparently quite normal.

The *Cerebral Cortex* (left frontal convolution) and the *Crus Cerebri* appeared quite healthy.

The *Medulla Oblongata* presented the following pathological changes:—

(1) The fasciculus gracilis (Goll's tract) was generally sclerosed.

(2) Sclerosis affected the restiform body, and the cells were only faintly stained.

(3) Gowers' tract took on the stain badly and the axis cylinders appeared to be atrophied.

(4) The fillet fibres were normal anteriorly, but posteriorly there was general sclerosis with isolated patches of nerve-fibres which were undoubtedly degenerated.

The Sciatic nerve in cross sections presented changes which were markedly pathological. The nerve-fibres were closely packed together and pressed into masses by an increased deposit of fibrous tissue, which in places was broken down into debris-like accumulations in which no nerve fibres were observed at all. Some fibres were devoid of axis cylinders altogether; in others the axis was swollen. The empty appearance presented by the sheaths in places was very characteristic and looked like a honeycomb arrangement.

There was no uniformity in the size of the nerve-fibres; some were large and some were small. Whether the latter were due to simple pressure or whether they are actually embryonal structures is doubtful.

In sections stained with hæmatoxylin (Weigert) there were patches of the section which failed to take on the stain at all, consisting of very small nerve-fibrils, much atrophied and evidently much compressed by replacing fibrous tissue. These patches of sclerosis were all the more evident for the other portions of the section took on the stain admirably.

REMARKS.

In BRAIN (1898, page 72) there is an interesting clinical record of several cases of Friedreich's ataxy by Dr. Mackie Whyte, and in the same volume at page 438 there is a very thorough article on the pathology of this disease by Dr. H. Mackay—both papers being supplemented by an extensive bibliography.

It may here be of interest to review the symptoms presented by our case which justified our diagnosis.

(1) Two cases occurred in the same family. The occurrence of paralysis, of a progressive type, in more than one member of a family during childhood should make us suspect this disease, and lead us to further observation.

(2) The disease commenced in both our cases immediately after a post-scarlatinal chill, and most observers hold that, as an exciting factor, some acute disease, especially if accompanied by fever, is generally the direct cause of the paralysis, the predisposing cause, of course, being a tendency to degeneration of the neuron.

(3) The first symptom noticed in our cases was a difficulty in walking—the ataxia being progressive in character.

(4) There was a rapid change in the power of articulation, so that, early in the history of the disease, the power of speech became well nigh abolished.

(5) As a later symptom in the progress of the disease, ataxia of the hands occurred; and hyper-extension of the fingers and toes, especially of the right hand and foot, is a prominent symptom in the case of Charlie.

Main en griffe—claw-like hand—is a condition which all along has been absent in our cases, although it is described as occurring in several of Dr. Mackie Whyte's patients.

(6) Athetoid movements of the fingers and toes, especially when the attention is diverted, were well marked in the case of the girl, although they do not form so prominent a symptom in the boy.

(7) An utter helplessness of the body exists in the case of the boy, and was very marked in the girl. The patient has to be carried about from place to place; he is quite unable to support himself unless when he is seated, and then he lies back on the chair, or else when he is lying down in bed. His head falls from side to side as if the neck muscles were quite unable to support it.

(8) There is marked dorsal spinal curvature—lateral; the apex of the deformed spinal column reaching the posterior border of the scapula in the case of the boy, and scoliosis was just as marked and in the same position in the girl.

(9) Talipes equino-varus, especially of the right foot, occurs in the case of the boy. Double talipes equino-varus existed in the case of the girl.

(10) Nystagmus was a very marked symptom in the girl. It is only slightly evident in the boy.

(11) The visceral reflexes in micturition and deglutition were affected; the former slightly in the boy's case; the latter, to a marked degree, towards the end of the girl's life.

(12) There existed marked incoordination in both cases. They were unable to touch the point of the nose without

first some hovering and hesitation of the finger in its progress.

(13) Electrical reactions in the case of the boy, so far as they go, were practically normal.

(14) There was an entire absence of the knee-jerk, and most of the deep reflexes were either impaired or abolished altogether.

(15) Finally, the intellectual faculties in both cases were only slightly impaired; no more, indeed, than might be expected in any case of chronic paralysis.

One curious fact about one case is that although the motor symptoms are mostly bilateral, still the ataxia seems more marked on the right side than on the left, and it may be that the spinal curvature pointing to the left side is due to the more advanced paresis of the right arm as compared to the left. This explanation is given by Mackie Whyte.

As regards the differential diagnosis of Friedreich's paralysis it is to be noted that there is a close similarity between it and hereditary cerebellar ataxia. Marie's theory, as pointed out by Whyte, may be correct, that the same degenerative process affects the spinal cord in the one case and the cerebellum in the other.

From disseminated sclerosis and locomotor ataxy, Friedreich's disease may be distinguished clinically by its family character, its occurrence in childhood, and an absence of many of the symptoms characteristic of these diseases, such as jerky "intention" tremors and ocular affections in disseminated sclerosis, and the "girdle" pain, and various crises—gastric and otherwise—of locomotor ataxy.

It should not be forgotten that cases have been described, among other observers by Friedreich himself, in which the symptoms indicated disease of a hybrid nature. Such cases are, however, the exception, and do not disprove the fact there is a group of symptoms, affecting the motor nervous system and characterised by distinct lesions in the spinal cord, medulla and nerves, forming a distinct entity and to which Friedreich gave his name.

A consideration of the pathogenesis of Friedreich's disease is both interesting and instructive. Dr. Pitt (*Guy's Hospital*

Report, 1886-7) puts forward the theory that this disease, with its imperfect development of the spinal cord, is vascular in its origin, because—

(1) The posterior columns of the cord are the most vascular, and here the sclerosis is most marked.

(2) Vascular lesions have been described in some cases, and the disease is always aggravated by acute affections.

(3) Development of the cord begins in the most vascular regions, so that degeneration attacks, by preference, the latest developed parts.

(4) The sclerosed regions correspond to tracts, the filaments of which are covered by myeline after the fifth month of fœtal life.

(5) The symptoms become more marked at puberty, which is accompanied, as we know, by great vascular modifications in the organism.

And, again, Guizetti (*Reforma Medica*, June, 1893) maintains that Friedreich's disease "depends upon a congenital predisposition, in consequence of which, in the early years of life, certain systems of fibres and nerve-cells undergo a process of progressive atrophy, and that the process is independent of any contributory effect from vascular alterations."

Mackay describes these "certain systems of fibres and cells" quoted by Guizetti, as those which are latent to undergo medullation and those which are functionally related to the postero-internal tract.

The entire absence of disease of the coats of vessels in our case, although they were mostly full of blood, disproves somewhat Pitt's theory as to a vascular origin. On the other hand, while there may be a predisposing factor in developmental arrest and congenital malformation of the spinal cord, it is interesting to note that, in the majority of cases recorded, the first symptoms of the disease observed have followed upon some acute febrile state of the system, in which it is reasonable to suppose a congested condition of the spinal cord vessels existed.

We are disposed to favour both theories, in part. That the disease is due to an arrest in the development of certain strands of nerve-fibres and cells in the cord, the results of which are not evident until some intercurrent acute affection occurs, sums up briefly our present ideas of the pathogenesis of Friedreich's paralysis.

It remains for us briefly to discuss the prognosis and treatment. As regards the former, Friedreich's ataxia is progressive and chronic, many cases living, according to Ladame (BRAIN, 1890, p. 525), to upwards of 40 years of age.

Although intermissions are said to have occurred, yet in no case has a recovery been reported. The disease either kills by itself or some intercurrent affection may cause a fatal termination.

As in all spinal cord affections, the duration of life depends, to a marked extent, upon the character of the nursing, and it is interesting to note here the almost entire absence of any tendency to bedsores occurring. Our patient sits day after day on the same chair, and yet his skin remains unbroken, and the girl's slight bedsore was due more to emaciation than to any trophic affection.

With such a prognosis little need be said as to the treatment of Friedreich's ataxy. Charcot recommended "suspension" with apparent benefit; Zalludowski gave temporary relief to a little girl by means of massage to the limbs, and Mackie Whyte recommends exercise and an education of the functions of those muscles which tend to become affected. Our patient says that he feels very much better after the use of the continuous current. Drugs seem to have little influence in the arrest of symptoms, and the most we can do for cases is to nurse them carefully and attempt to combat the graver symptoms as they occur.

In conclusion, we would express our thanks to Dr. le Doux, of the Bacteriological Institute, for his kindness in making sections and photomicrographs of various parts of the nervous system which illustrate this paper.

A CASE OF SYPHILITIC SPINAL LEPTO-MENINGITIS WITH COMBINED SCLEROSIS.

BY A. F. SHOYER, M.B., B.C.CAMB.

Assistant Medical Officer and Pathologist, County Asylum, Lancaster; late Resident Medical Assistant, Royal Infirmary, Dundee.

THE patient in question first came under observation in the Royal Infirmary, Dundee, where he was under the care of Dr. Mackie Whyte, and afterwards in the Dundee Poorhouse Hospital, again under the care of Dr. Whyte, to whom I am indebted for permission to publish the notes of the case and for the material for microscopic examination. Altogether the patient was under observation from October, 1897, until February, 1900, when he died. The notes of his case while in the Royal Infirmary I give verbatim, as nothing of them is superfluous.

R. L., aged 61, labourer, was admitted to the Royal Infirmary, Dundee, on October 7, 1897.

Complaint.—(1) Pain in right side of chest; (2) shooting pains in left arm and leg.

Duration.—(1) One week; (2) two years.

Previous history.—He has always been a strong healthy man, never having had any illness, except measles or scarlet fever. His work has been heavy, and has necessitated constant exposure to weather. He has suffered from "rheumatism" at times, but has not been subject to chest troubles. He has been a total abstainer for eight or nine months, but before that was a heavy drinker. He is a non-smoker. There is no history of venereal disease. Micturition has been more frequent for the last nine months, obliging him to get up three or four times at night.

Family history.—Negative. Married. Wife and six children healthy.

Present illness.—Two years ago, for no reason, a pain came on one night in his left heel; the foot and leg as far as the knee were swollen next day, but the swelling subsided without treatment and has never returned. The pains have continued more or less ever since, shooting sometimes from the heel up the leg, and up to the shoulder and down the arm, and sometimes in the reverse direction. Rubbing relieves the pain, which is severe enough to keep him awake at night. They have never affected the right side. In the left hand he often feels a sensation "like pins and needles." Walking is affected, as he never feels as if he were gripping the ground with his left foot. His memory has been failing for four or five years. The grasp of the left hand is weakened. A week ago a pain started in the right side of the chest, associated with a cough, worse at night, but relieved if he lies on his right side. He has lost three stone weight in four years. Suffers from frontal headache.

Physical examination.—He is a powerfully-built man of fairly good colour. Tongue clean and moist, not tremulous. Heart and lungs appear healthy.

There is tenderness all over the right side of the chest, extending as high as the third intercostal space, reaching to the middle line back and front, worst over the points of each of the intercostal nerves. There is tenderness in the right lumbar region and over the liver. No enlargement of liver or spleen. There is considerable loss of power in the left leg. Patellar and plantar reflexes exaggerated; no ankle clonus. Hyperæsthesia all down left leg, tenderness especially marked over the course of the sciatic nerve. No wasting or coldness, no ataxia; but the left leg is dragged when he walks, and he seems afraid to trust it. He says there is pain in the heel when walking, also that the ground is "not gripped properly." He can stand with his eyes shut, but cannot walk along a straight line. The grip of the left hand is very feeble. Dynamometer, right 30, left 10. There is hyperæsthesia of the left arm, and a feeling of pins and needles, especially in the hand. The pupils respond to light.

October 8. Urine: specific gravity 1006, acid, colourless, no albumen. The excess of sensitiveness is noticed on the scalp and face, as well as on the body, and is more marked on left side.

October 11. Sleeps well. Pain still severe in left side.

October 19. Patient exhibits a general condition of muscular weakness all over his body. His gait is tottering and shuffling, with very short steps, hardly raising his feet, but sliding them along. The left leg is distinctly the weaker. The arms are also very weak, the left more so. Dynamometer, right 30, left 20. He cannot protrude his tongue much further forward than his teeth. The movements of the eyes are good and the reactions of the pupils normal, but the pupils are small, and the right somewhat smaller than the left. There is a general cutaneous hyperæsthesia all over the body from head to foot, and this is more marked on the left side than on the right. There is ataxia of the upper extremities, possibly due to weakness. No ataxia of lower extremities can be demonstrated, and the positions of the legs seem fairly well appreciated. Besides the hyperæsthesia, no affection of sensation is to be found. The deep reflexes appear exaggerated, possibly due to hyperæsthesia, as on the left side a tap on the knee or wrist evokes an exclamation of pain, and sets the muscles into spasmodic clonic contractions. No ankle clonus. Plantar reflexes well marked. Abdominal reflex well marked on right side. Takes food well, but has great difficulty in feeding himself. Peculiar odour about patient's skin. Sphincters act efficiently.

November 1. Cannot open mouth above three-quarters of an inch. Gums soft, and bleed easily. Speech is wanting in force, and such as one would expect from the weakened muscular condition.

November 19. Sent to poorhouse.

His temperature was usually slightly above normal, sometimes reaching 100° and once 101°. Towards the end of his stay it became subnormal.

From the notes of his case while in the Poorhouse Hospital I extract the following :—

November 7, 1898. Temperature 97·4°. Urine: specific gravity 1026, acid, no albumen. Patient was in hospital ten days ago for pain in, and inability to move, his right knee. He was put under chloroform, but nothing could be made out; the case was supposed to be one of hysteria. He now returns, complaining of pain in, and inability to move, the right hip-joint. On examination, nothing is made out.

May 17, 1899. Continues in a very helpless state, rigidity being the chief characteristic. This is especially to be observed along the spinal column, right shoulder, right hip, knee, and to a less extent in the right elbow and left knee. No joint deformity can be made out, and there is no special muscular atrophy connected with any joint, although the whole muscular system is below par. Attempts at movement cause pain. He manages to feed himself in a slow and tremulous fashion with his right arm.

January 19, 1900. Rigidity increasing. The left elbow and shoulder are now both involved, and the jaw is stiff. Pain only present on movement.

February 10, 1900. The patient can now hardly speak; his lower jaw is rigid, and the left arm is kept close to the side. The shoulder is also rigid, movements in the elbow are slight, the right fingers show voluntary movement. The pectoral muscles are firmly contracted, the muscles of the abdomen rigid and board-like. Lumbar region is arched. Breathing entirely abdominal and shallow. Head is rigid, somewhat extended, and rotation is impossible. Temperature in rectum usually 95·8°, never more than 96°. Patient has not spoken for two days.

February 16, 1900. Eyes strongly deflected to right, pupils small, and the head is more retracted. He is quite apathetic. Body all over extremely cold. Thermometer in rectum registers 92°.

February 17. Died.

Diagnosis during life was chronic spinal meningitis.

SUMMARY OF SYMPTOMS.

The case ran a course of about four and a half years. The symptom first noticed was pain referred to the left heel, followed by shooting pains up or down the left side. There were also referred pains in the right side of the chest. Next came hyperæsthesia, not only cutaneous, but also muscular; for there was pain on using the muscles, and as result of the hyperæsthesia of the bladder we have frequency of micturition. There was muscular weakness and great loss of weight, and finally rigidity, which involved the muscles of respiration and led to death. These symptoms all point to thickening of the spinal membranes, with compression of the nerve-roots. They were not symmetrical on the two sides, and proceeded from below upwards. In addition there was ataxia of the upper limbs and doubtful spasticity of the lower, symptoms which imply involvement of the cord, and would be easily explained by the pathological changes actually found. No history of syphilis was obtained, but the changes in the arteries found and described below leave very little doubt as to this being the cause of the disease.

The very low temperature before death was no doubt due to deficient aeration.

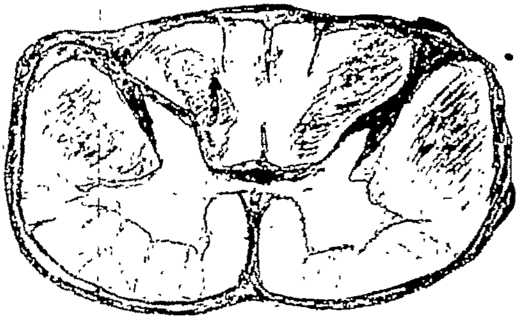

FIG. 1.

(*Semi-diagrammatic*).

Spinal Cord, Cervical region.

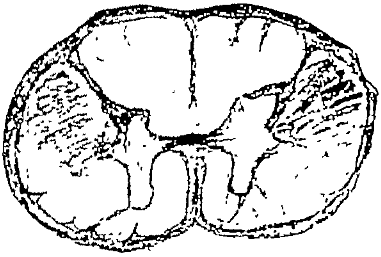

FIG. 2

(*Semi-diagrammatic*).

Spinal Cord, Dorsal region.

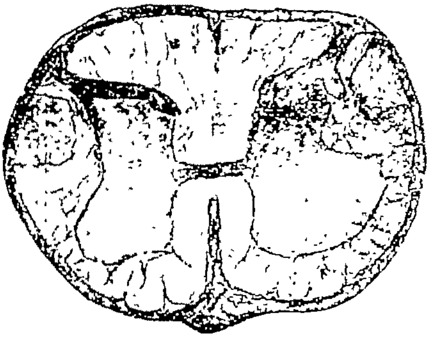

FIG. 3.

(*Semi-diagrammatic*).

Spinal Cord, Lumbar region.

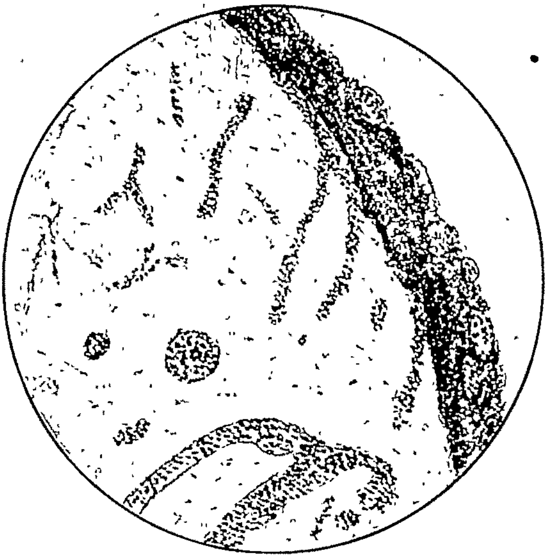

FIG. 4. × 50.
Periphery of Lateral Column in Cervical region.

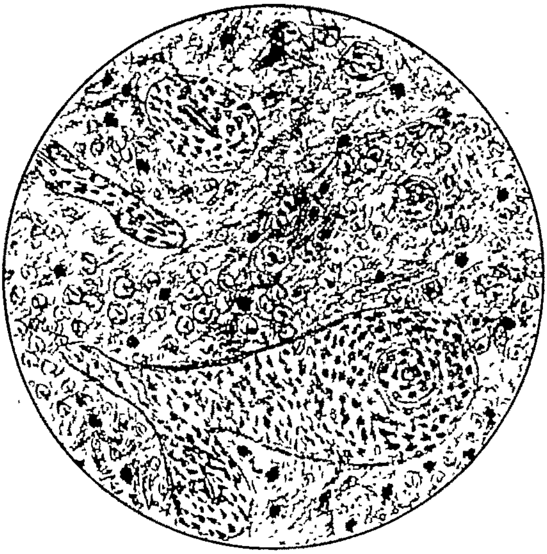

FIG. 5. × 200.
Some Arteries from Posterior Column in Cervical region.

The retraction of the head and deflection of the eyes just before death indicate some basal meningitis.

PATHOLOGY.

By the kindness of Dr. Mackie Whyte I received the brain and spinal cord.

The brain presented nothing abnormal to macroscopic examination, but as it had been in spirit, it was impossible to ascertain the presence or absence of basal meningitis. The spinal dura mater appeared quite normal, but the pia mater was tough, thick, and adherent firmly to the spinal cord, especially over the dorsal half of the circumference.

Preparations of the cord at about ten different levels showed more clearly this increase in thickness of the pia mater, and in addition sclerosis of both lateral columns throughout the length of the cord, involving both the direct cerebellar and the crossed pyramidal tracts. In the cervical region there is also sclerosis of the posterior columns, here even more marked than that of the lateral, and involving especially the postero-external tracts and the posterior root zones of Lissauer.

On close examination of the sclerosed columns, the most strikingly pathological condition is found in the arteries. These are in a condition of advanced arteritis, both endarteritis and periarteritis; the endothelial lining of the intima is proliferated until the lumen is extremely small or obliterated, while the media and adventitia are replaced by connective tissue crowded with nuclei. The arteries thus form almost solid columns, whose cross-sections take up a large proportion of the area of the section of the affected region, and contribute most to the naked-eye appearance of the sections.

These changes in the arteries are usually considered to be characteristic of syphilis; hence the title of this article.

Sections of the medulla at various levels show the pyramids along their whole length to be in the same condition as the lateral columns of the spinal cord. This is more remarkable than appears at first sight, for while regarding the lesions in the cord alone we could assume the disease to originate in the arteries, for the districts affected are all served by the posterior spinal arteries and their branches, the anterior spinal artery and its area of supply being healthy, or but slightly affected. But when we come to the medulla we find the pyramids supplied by the anterior spinal artery are the only diseased parts, and that here the anterior spinal artery is the diseased artery, the others being healthy. This almost forces the conclusion that the origin of the disease is really in the motor tracts. As a corollary to this we might assume that the lepto-meningitis, to which the symptoms were due, was secondary to the arterial disease, and therefore to the disease of the motor tracts.

Sections of the pons showed an increase of nuclei in the coats of the arteries supplying the pyramidal fibres, but nothing approaching the condition lower down.

Sections of the cortex from the motor region showed healthy arteries and abundant healthy pyramidal cells, while tangential fibres were well developed. The nerve-cells in the gray matter of the spinal cord were healthy.

A CASE OF ERYSIPELAS OF THE SCALP WITH A REMARKABLE NERVOUS SEQUELA.

BY F. J. POYNTON, M.D.

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THE HISTORY OF THE CASE.

THE patient, a youth, aged 17, was first an inmate of St. Mary's Hospital under Dr. Cheadle, later he was transferred to the Chelsea and Willesden Infirmaries, and finally was admitted under Sir William Gowers to the National Hospital, Queen Square.

I am indebted to the kindness of Dr. Cheadle and Sir William Gowers for leave to make use of the notes that were taken while he was under their care, and to the courtesy of the Superintendents at the two Infirmaries for information as to his condition during that period of his illness.

The duration of the illness was a long one, extending from October 20, 1897, to April 14, 1899, and it so happened that I was able to keep in touch with him throughout, and to follow his subsequent history.

E. R., aged 17, was admitted to St. Mary's Hospital upon October 20, 1897, complaining of giddiness and pain in his head and back. The illness had commenced quite suddenly three days previously, and he took to his bed the day before admission. The only fact that suggested a cause was that his mother had recently returned from a London fever hospital convalescent from an attack of scarlet fever. This raised the suspicion that the dwelling house was insanitary. He was very ill, the temperature 104°, pulse 102 per minute, respirations 30. The tongue was thickly coated, and the bowels were constipated. Though quite sensible the least movement pained him, and he lay curled up on his side, with the eyelids closed. The aspect was pale and anxious, and the patient complained bitterly of pain in the head, in addition to which there was occasional vomiting. Some enlarged glands were detected behind the left ear, and the veins over the left side of the forehead were prominent and distended. There was no membrane upon the throat, no scarlet redness or rash upon the chest, nor did the condition of the other systems throw any light upon the condition.

Later in the day exquisite tenderness developed over the head; this was more marked upon the left side than the right, and with it there was also some tenderness down the left side of the neck. There was slight double optic neuritis, but there were no ocular palsies and the pupils reacted to light and accommodation. The tympanic membranes were quite healthy. The next day the symptoms remained much the same, except that the tenderness over the head became more general. The knee-jerks were tested several times and were never obtained. Upon October 23 a dull flush appeared upon the forehead spreading from the hair line, and the skin in this region was œdematous. The next day it was clear that he was suffering from a severe attack of erysipelas of the scalp, and from blood obtained from the spreading margin I obtained a pure culture of streptococci. Antistreptococcin in 10 cc. doses was injected upon the 26th and 27th. At this time the patient's condition was perilous. His face was now greatly swollen, the mental condition dull, and the temperature oscillating between 104° and 106°. Upon October 29 there was a crisis, the temperature fell to 99°, and desquamation commenced upon the face. After a slight relapse the temperature fell to normal upon November 4. From this date there was a steady improvement, and by the end of the month he was able to sit upon a chair, and in December was able to walk and was apparently convalescent.

In the second week of this month a curious change took place: his appetite failed and the bowels became constipated, the temperature did not, however, rise. He became very irritable, was morose even to his friends and complained that his legs gave way suddenly letting him fall. There was no loss of consciousness. The change was so marked that he was compelled to take to his bed. Headache and vomiting now resumed, and at times he was very drowsy. The knee-jerks were now active and presented that peculiar oscillation which is seen not infrequently in chorea: there was no ankle clonus. In the middle of January there were sudden unexplained rises of temperature to 101°, the bowels remained obstinately constipated, and for the first time there was retention of urine. Both lower extremities were weak and he was unable to support his weight. The grasp was also weak on both sides. Sensation was blunted over the lower extremities, but not lost. The respiration was sighing. Pain had been complained of in the back, but it was not accurately localised, and there was no localised tenderness. Upon January 20 there was incontinence of urine. He remained in the hospital until April in much the same condition, completely bedridden, with retention varied with incontinence of urine. The lower limbs, though they could not be raised from the bed, were not spastic, nor had they wasted to any marked extent.

After leaving the hospital he was an inmate of the Willesden and Chelsea Infirmaries. Through the courtesy of the Superintendents information was obtained of his condition during this period, of which the following is a summary. He remained in very much the same helpless state, getting at first if anything rather worse, for the legs became weaker and the incontinence of urine more frequent, and in addition there was an occasional girdle pain. Towards the end of 1898 he began to improve, the legs becoming stronger.

Upon March 1, 1899, he was admitted to the National Hospital under Sir William Gowers, and for the following notes I am indebted to Dr. Edwin Bramwell, at that time the junior house physician. He was thin, his manner depressed, but intelligence good. There was some lateral nystagmus. The fingers moved well and equally, but there was some slight weakness of the muscles of the arm and forearm upon the right side. The movements of the upper limbs were shaky and suggestive of the intention tremor of disseminated sclerosis. The handwriting was very shaky.

The abdominal muscles were weak. He was unable to raise his legs off the bed and no movement could be made at the knee or ankle joint, yet slight interrupted movements of the muscles were noticeable when movement was attempted. The toes were moved rhythmically some three to four seconds after the suggestion was made. The legs were thin; there was no rigidity. Sensation was also altered, thus there was loss of muscular sense in the right arm, and diminished sensibility to touch and pain up to the fifth rib, and analgesia to the seventh. The upper extremities were not anæsthetic. The knee-jerks were increased, but there was no ankle clonus. The plantar reflex was absent at first, but later there was a flexor response. Incontinence of urine was the rule varied by occasional retention, and the patient stated that he was sometimes aware when the urine was passed, but had no control. The urine contained pus. The bowels were constipated, but sometimes moved without his knowledge. There were no bed sores. The bladder was washed out daily, and strong faradism applied to the legs. Improvement was almost immediate. Upon March 12 there was an unaccountable rise of temperature to 103°. Upon the 18th he was so much better that the incontinence of urine had all but ceased. Upon the 21st he had complete control and could actually walk a few steps. He left the hospital upon April 14 and could then walk a mile.

After leaving the National Hospital he went to a convalescent home and returned quite strong and well. Later in the year he showed himself to Dr. Cheadle in capital health. In the autumn of 1899, he came to see me in answer to a note, and was then in robust health. His appearance had much altered, he was stout with a good colour, quite strong on his legs and with no urinary troubles at all. The knee-jerks were natural.

Thus after an illness extending from October 20, 1897, to April 14, 1899, during the greater part of which time the patient had been bedridden, there resulted a complete recovery.

This case presents several points of interest both practical and theoretical. From the point of view of treatment, it furnishes a proof of the value of persistent and assiduous care in a condition which, on more than one occasion, looked anything but promising. If this patient had gone home early in the illness, there is little doubt that the cystitis, which even with care did eventually make its appearance, would have arisen earlier, have been more severe and would have caused irreparable damage to the kidneys..

The use of the antistreptococcic serum was rapidly followed by the termination of the acute attack, but from personal observation I do not feel convinced that this was anything but the natural crisis which would have occurred irrespective of the use of the serum.

The rapid success attending the use of strong faradism was a signal proof of the value of this method in suitably chosen conditions.

The injury to the mechanism of the bladder was another remarkable feature of this case. For a long while there was almost complete incontinence, and yet the result was recovery and complete control. The symptom-complex at the time that there was incontinence was unusual, for the lower extremities, though weak, had not wasted more than could be accounted for by disease, and the knee-jerks were more than usually active. On the other hand, when there was retention of urine there was never any rigidity of the lower limbs, nor ankle clonus. The condition was a marked contrast to that intensely organic affection of the cord, anterior poliomyelitis, in which with severe flaccid paralysis of the lower extremities the sphincters as a rule escape.

How much of this illness was functional, in the sense that it was dependent upon lack of will power on the part of the patient, is a difficult question to answer. The severe implication of the bladder certainly seemed to point to an organic lesion of some severity, and to a bad prognosis, and even when recovery occurred, there still remained the doubt as to whether this was only temporary, and not, as has proved to be the case, permanent.

The explanation of the cause of this sequela is to a great extent a matter of hypothesis. It is not at all likely that the condition could have been produced by the injections of antitoxin, though we cannot lose sight of the fact that this serum was used upon two occasions.

The stages through which the illness passed were firstly that of the acute local infection, to which no doubt the severe cerebral symptoms at the outset were due; then a period of improvement, which almost reached the point of recovery, next a gradual mental deterioration rapidly followed by the appearance of paralytic symptoms; fourthly a prolonged stationary period, and lastly, a stage of rapid recovery.

The primary infection may have damaged the brain considerably, and the gradual development of the new train of symptoms after a latent interval resembles in this respect that occasional sequel of a brain shock or injury—chronic traumatic neurasthenia. In this case the local infection would take the place of the more gross mechanical injury.

It is also possible that this sequela was the direct result of the virus of the erysipelas which persisted in an attenuated or altered state. Much light has been thrown by neurologists upon the occurrence of nervous symptoms as an after-result, proximate or remote, of acute infections, and the question is one of far-reaching importance. Now that almost each year we are confronted with new micro-organisms, each looked upon as the specific cause of some particular disease, the complexity of the bacteriological problem is becoming very apparent. A survey of the group of coccal infections well illustrates this difficulty. There are the streptococci and the staphylococci, the diplococci of pneumonia and rheumatic fever, of scarlet fever and cerebro-spinal meningitis. There is the gonococcus and the diplococcus described by Still in posterior basic meningitis. Morphology alone can hardly be expected to distinguish them, much less any method of staining, and it seems that chemical pathology by a study of the poisons that are produced and the reactions that are called out will eventually afford the means of differentiating these processes.

Nervous disturbances resulting from infections are of such interest because they are to some extent a clinical expression of these subtle chemical processes. If this case be looked upon as an example of this kind then it is a fortunate one, because the organism was isolated at the commencement of the illness and the patient under observation throughout its entire course.

Reviews.

A Treatise on Mental Diseases, based upon the Lecture Course at the Johns Hopkins University, 1899, *and Designed for the Use of Practitioners and Students of Medicine.* By HENRY J. BERKLEY, M.D., *Clinical Professor of Psychiatry, the Johns Hopkins University, Chief Visiting Physician to the City Insane Asylum, Baltimore.* With Frontispiece, Lithographic Plates and Illustrations in the Text. (New York: D. Appleton & Company, 1900.)

THE object of the author of this treatise has been to supply English medical literature with "a comprehensive, practical work on Mental Diseases," and the production of a most admirable text-book for practitioners and students is the result. Both the wide view of the subject adopted and the absence from the work of such a chapter on the medico-legal aspects of insanity as would give it a more or less local character, fit it for the large class of readers for whom it is intended, and the general clearness of the descriptions and the interesting manner in which every branch of the subject is treated cannot be too highly praised.

The book opens with a short and lucid description of the general anatomy and histology of the cerebrum so far as this has a direct bearing on the study of mental diseases, and the absence of unnecessary details of structure has enabled the author to compress this portion of the subject into fifty pages of highly readable matter. The general structure of the cortical neurones and of the neuroglia is beautifully illustrated by drawings of Golgi preparations, and excellent diagrams of the nerve elements and the lympathic circulation of the cortex are introduced. The finer structure of the nerve-cell as shown by the Nissl method is clearly described and well illustrated, and a satisfactory account is given of the cell layers and association fibres of the cerebrum.

This part of the work is followed by a description of the general and special pathology of mental disease. In the introduction to this part the author lays especial stress on an inherited predisposition to disturbances of function and to premature retrogression of the cortical cells, the development of mental disease being associated with original defective construction of the neurone, vascular lesions occurring at the retrogressive period of life, or general toxic conditions. Whilst the description of the gross pathological anatomy of the cerebrum is short and only moderately good, that of the experimental pathology of the nerve-cell is excellent. Very clear accounts of the changes in the cell protoplasm in primary and secondary degeneration are given, as is also a description of the author's own researches on the nuclear changes due to the action of ricin and alcohol on the cortical nerve-cell, this last being of especial value owing to the relatively small amount of attention which is usually paid to this subject. The writer contrasts the excellent results obtained from experimental lesions with those in the human subject, and is inclined to lay more stress on nuclear changes than on changes in the Nissl-bodies. He also considers, from the experimental standpoint, that Golgi preparations are trustworthy as regards morbid changes, and his views in this matter, though contrary to those generally accepted, are entitled to respect in that they are founded on certain personal investigations which he briefly refers to. He, however, discounts the value of the Golgi method when applied to the human subject owing to the presence of *post-mortem* products, &c. Unfortunately the morbid changes in nerve-fibres are barely referred to, but an excellent description of the pathology of the cerebral vessels and of syphilitic vascular lesions is given. The section closes with a short illustrated description of abnormalities of the basal arteries. The illustrations in this section are only moderately good, and at least one or two cells showing Nissl changes might well have been introduced. The literature also is very incomplete, and shows an entire absence of reference to English authors, the only two writers in English referred to being Americans. This may partially explain the views of the author on the subject of cell changes in the human subject. The same remark with reference to the literature might be made in the case of several succeeding sections of the work.

In the first group of mental disorders treated, namely, "the idiopathic insanities without ascertainable alteration of the brain substance," is included a description, occupying only three pages of letterpress, of secondary dementia. It is to say the least, somewhat remarkable that a group which includes probably half the total inmates of asylums should be dealt with so very briefly, and also that a condition which in its later stages presents gross pathological appearances second in degree to those of general paralysis alone, should be placed under the heading of idiopathic insanity without ascertainable pathology. This contradiction is emphasised by the presence in the article of a cut illustrating marked dilatation of the lateral ventricles in a case of chronic mania, which, however, is not referred to in the text.

Under group 2 are included the insanities consecutive to demonstrable organic lesions. The account begins with a description of "progressive paralysis of the insane." The author lays the usual stress on syphilis as the pre-eminent ætiological factor in the disease, and it is satisfactory to note that he includes heredity as the other important predisposing cause, here agreeing with Näcke, who found a hereditary history of insanity in 50 per cent. of his cases. The symptomatology of the disease is good and is followed by an excellent summary of the more important facts. The author refers shortly both here and in a later chapter to the juvenile form of the disease. In the account of the pathology of general paralysis the writer accepts the theory that the disease is of vascular origin and usually caused by the direct or indirect effects of syphilis on the cortical vessels, and considers the changes in the nerve-cells to be secondary and indefinite. "The Golgi and Nissl methods have unfortunately added but little to our knowledge of the conditions of the neurone in paresis." The description of the pathology of the disease whilst as a whole good rather partakes of the nature of an *ex parte* statement of facts, and in this agrees with the author's general tendency to ascribe the majority of the insanities of this group to vascular lesions of primary origin, or due to the action of toxines, with secondary neuronic changes. This is curious when one considers the high metabolism and the unstable nature of the neurone, and the fact that, except in senile cases, the author acknowledges the influence of heredity in inducing defective construction and imperfect vitality of this structure. In the article on syphilitic insanity the author makes the rather surprising statement that this disease "is quite as frequent or even more common than general paresis and tabes dorsalis," and then explains this by stating that "all cases under 40 years of age that are hemiplegic or monoplegic, with temporary or permanent mental disturbance, and in whom there are no indications of renal or cardiac lesion, are with us ascribed to syphilis, as are likewise many of the obscure dementias that occur between the 25th and 40th years." The pathology of syphilitic insanity is described in half-a-dozen lines, which is very unsatisfactory in view of the above statement. In the article "psychoses of old age," quite a lengthy description, occupying nine pages, is given of the pathology of the disease and especially of senile vascular changes in the cerebral vessels, and this is followed by a short account of organic dementia. The clinical description of the alcoholic insanities is particularly good, but the pathology given consists almost entirely of an account of the author's researches on alcohol poisoning in rabbits.

In the fourth section, idiocy, cretinism and imbecility are treated in an article which, both clinically and pathologically, is much above the average, and in the description of imbecility the author refers to the frequency of insanity, which often ends rapidly in dementia, in the higher class of imbeciles. In this section is included an excellent article on cranial measurements and the stigmata of degeneration, with an illustrated description of the author's observations on abnormalities of the palate.

Whilst the general descriptions in the clinical section are good it is difficult to see why groups 1 and 3 are separated from one another, and it is remarkable that no reference, even under another name, is made to hypochondriasis, which is such a common symptom-complex in both the sane and the insane. The clinical section would probably have been improved by an introduction dealing with those details of psychology which have a special bearing on the phenomena of mental disease, and with the symptoms of mental derangement considered from a psychological standpoint, for as the subject is treated in the present work the student will necessarily gain the impression that insanity is a series of diseases instead of a series of symptom-complexes which have a common origin in lesions of those parts of the nervous system concerned with the processes of thought.

In text-books written by such an acknowledged authority as the author of this work, it is no doubt desirable that the individuality and opinions of the writer should have all due attention paid to them. It seems a pity, however, that in so many works, as in the present one, so much stress should be laid on individual opinion, and so little attention paid to the views of other authorities, for a text-book should be essentially a statement of known facts and theories rather than a collected edition of the researches and views of the author.

The index is excellent, and the illustrations as a whole are very good, especially the photographs of patients. Here and there words unfamiliar to the English reader such as "obstipation," "arrestation" and "coprolalia" are to be met with, and occasionally, according to American custom, the words are curiously divided, as for example, "cellu-lifugally," "ma-niac," and "arterioscle-rosis."

The book as a whole, apart from the blemishes to which attention has been drawn, is an excellent one, and should not only be most useful to the wide class of readers for whose use it is written, but will also be found to contain a considerable amount of matter of interest to the alienist.

J. S. Bolton.

A Text-Book of Pathology in Relation to Mental Diseases. By W. Ford Robertson, M.D., *Pathologist to the Scottish Asylums; formerly Pathologist to the Royal Edinburgh Asylum.* Illustrated with 16 lithographic plates in black and in colours, and 13 engravings. (Edinburgh: William F. Clay, 18, Teviot Place, 1900.)

It is doubtful whether the publication of a work of this kind is desirable in the present early and rapidly-advancing state of our knowledge of the pathology of the cortical neurone, as such a text-book is necessarily almost out of date by the time it reaches the hands of its readers. Few neuro-pathologists, however, are better qualified for such a task than the author of the present work, for, as he states in the Preface, he has devoted the past seven years to researches in this subject with a definite view to their final publication in book form. The work is consequently to a large extent, and especially in the case of some of the more valuable chapters, a republication of the author's previous papers. Of the twenty-nine plates, for example, which the work contains, only eight are new, the remaining twenty-one having been previously published by the author either alone or in conjunction with others. This fact, whilst not in any sense decreasing the value of the book, tends to throw into undue prominence the special researches which have been carried out by the author.

It is impossible to too highly praise the chapters on hæmatoma auris, the morbid conditions of the dura mater and pia-arachnoid, and the structure and morbid conditions of the neuroglia. In the chapter on hæmatoma auris the literature is fully referred to, and a well-balanced criticism is followed by an excellent summary of conclusions. In that on the dura mater the minute description of the degenerative conditions of this membrane and their relation to the formation of sub-dural false membranes is especially good, although the numerous illustrations are very diagrammatic, and do not reach the high standard of the description. In the excellent account of the normal structure of the pia-arachnoid, the author lays stress on the virtual absence of capillaries from this membrane, which is a fact hitherto not generally recognised. The chapter on the intracranial blood-vessels and the special histological characteristics of the intracerebral capillaries is also good. That on the neuroglia is, however, probably the most valuable chapter in the book, and contains the first complete account of the author's researches on this subject. He strongly supports the view recently formulated by certain authors, and especially by Capobianco and Fragnito from their rearches on the developing chick, that part of the neuroglia is mesoblastic in origin. The special characters of the two varieties of neuroglial cells are minutely described. Those of epiblastic origin or "neuroglia" are "special highly-branched cells, which vary greatly in size, and in the number and arrangement of their processes. . . . Their protoplasm . . . has become in greater part differentiated into a denser substance, which forms fine fibres. . . . Many of them are attached to the adventitia of the vessels either directly or by means of fan-shaped expansions." The cells of mesoblastic origin, or "mesoglia," are "probably about equally numerous, . . . have processes which are composed of the same substance as the cell body, and are generally markedly dendritic. Moreover, they have no connections with the vessels or other structures."

The chapter on the morbid conditions of the nerve-cell is mainly an amplification of the author's previously published critical digest. Whilst it is an excellent account of our present knowledge of the general pathology of the nerve-cell, a more minute description of the various types of primary cell degeneration might have been introduced, and the illustrations on plates xxvii. and xxviii. are not only unsatisfactory, but not a single typical example of a cell undergoing acute degeneration is shown. There is also no adequate description of or discussion on the morbid conditions of medullated nerve-fibres.

In chapter XIV. is given a brief account of the pathology of certain of the clinical types of mental disease. The author throughout inclines to accept the toxic or auto-toxic theory of the pathogenesis of the insanities (excluding imbecility) and lays relatively little stress on hereditary influences. He criticises Mott's advocacy of the theory of Tuczek that general paralysis is a primary degeneration of the nervous elements of the cerebral cortex, and accepts the toxic theory of Angiolella, giving an excellent description of the lesions of the cerebral capillaries in this disease. He does not however satisfactorily show that these lesions may not equally result from toxins derived from neuronic degeneration, and finds it "difficult to understand why the capillaries of the frontal and central convolutions should be specially liable to involvement." In his explanation of this he omits any reference to the anatomical arrangement of the veins of the fronto-parietal region, on which stress is laid by Mott, and to the fact that this region is the part of the brain most affected in ordinary insanity.

Chapter XII., on the cerebral circulation, is the least satisfactory part of the book. The author in it severely criticises the researches of Leonard Hill on the physiology and pathology of the cerebral circulation. In his attempted refutation of the Monro-Kellie doctrine of the incompressibility of the brain, he confuses compressibility with elasticity (pp. 293-4), and as a result denies Hill's statement that "the volume of the blood in the brain is in all physiological conditions but slightly variable." He then shows how, if Hill's facts be *not* accepted, the mechanism of the intracranial circulation can be explained by alterations in the amount of lymph present in the intracerebral lymphatics. He also accepts Cavazzani's statement (1892) that the cervical sympathetic contains vaso-constrictor and vaso-dilator nerves for the brain, in preference to those of Hill (1896 and 1899) that "evidence is not forthcoming of the existence of any local vaso-motor mechanism," and that "the cerebral circulation is controlled by the vaso-motor centre acting on the splanchnic area." In view of the author's denial of the Monro-Kellie doctrine it is not surprising to find that true œdema of the brain depends on conditions "the exact nature of which must still be regarded as obscure." Whilst the opinions of the author on this subject are entitled to consideration it is probable that readers will consider this chapter to be unsatisfactory, and it is to be hoped that it will be modified in a future edition of the work.

In minor details the book presents shortcomings of less importance. In the chapter on histological methods, for example, whilst the methods or modifications of methods preferred by or originated by the author are fully described, no description of the Weigert-Pal and Nissl methods is given. It is also to be regretted that the subjects which have not received the special attention of the author are so briefly referred to. Taken as a whole, however, the book is well written, the general arrangement is satisfactory, and the index, though not copious, is good. The work is a valuable contribution to neuro-pathology and will be of considerable assistance to the original workers for whose use it is intended by the author.

J. S. BOLTON.

Die Vasomotorisch-trophischen Neurosen. Dr. RICHARD CASSIRER. Karger, Berlin, 1901.

UNDER this title Dr. Cassirer, in a monograph of 600 pages, gives us an exhaustive study of such diseases as acroparæsthesia, erythromelalgia, Raynaud's disease, scleroderma, angioneurotic œdema and multiple gangrene.

As a fitting introduction to the work the first chapter of 100 pages is devoted to the physiology and anatomy of the various nerve-centres and tracts directly associated with the vasomotor, secretory and trophic functions of the nervous system. The several diseases are then considered separately in the following chapters.

Dr. Cassirer, in addition to his own observations made at Professor Oppenheim's Nerve Clinic in Berlin, has collected together a vast amount of information from other sources, and consequently he is able to tell us nearly everything of importance that is known of the ætiology, symptoms, pathology and treatment of these diseases.

The chapter on Raynaud's disease in particular is most complete and speaks to the thorough way in which the author has set about his laborious task.

Throughout the work numerous cases are considered in detail in order to show how intimately these diseases are related, and in the last chapter a brief summary is given of the author's conclusions, together with a suggestive table which shows the leading symptoms of these neuroses.

Vasomotor disturbances, as represented by local syncope, asphyxia and hyperæmia, are the essential changes which underlie all these conditions, and around these are grouped in various ways sensory, secretory and trophic disturbances.

The marked prominence of any one of these local disturbances changes the clinical type of the disease and thus produces a clinical variety which stands intermediate between one disease and another.

Raynaud's disease, on account of its multiplicity of symptoms, is placed as the centre around which the other diseases are grouped.

In this disease vasomotor, sensory, secretory and trophic disturbances are all represented in varying degree in different cases.

If, however, these disturbances become more marked in one direction we have a change in the clinical picture.

A case of Raynaud's disease may, for instance, show trophic changes in the fingers and toes identical with those seen in sclerodactylie.

Sclerodactylie again is sometimes associated with ordinary diffuse or circumscribed scleroderma, so that we have a clinical series of cases starting from Raynaud's disease and ending in scleroderma.

Again, if hyperæmia is a marked symptom in Raynaud's disease and other symptoms stand in the background we approach the condition of erythromelalgia.

In the same way the prominence of gangrene causes the clinical picture to approach that of multiple gangrene. Such examples briefly show the direction in which Dr. Cassirer formulates his conclusions.

An extensive bibliography, so necessary in these days, helps to complete a work which cannot fail to be an important book of reference to those who may be studying this interesting group of diseases.

E. Stainer.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

AT the CLINICAL MEETING held at 11, Chandos Street, on Thursday, March 21, at 8.30 p.m., the following cases were shown:—

(I.) Dr. F. E. BATTEN.—*Four cases of congenital ataxia in children.*

(1) F. W., aged 5, was first seen in July, 1900. He was the third of three children, of whom the first two were quite healthy. The labour was natural but the mother was not in good health while carrying the child. He learnt to talk at a year old. The nystagmus which is present has been noted since birth, and the squint a few months after birth. He has never learnt to walk.

When attempting to stand or walk, the child was very ataxic; there was marked incoordination of the hands, constant nystagmus, and the articulation was very defective. There was no rigidity of the legs, and no pes cavus or spinal curvature. The knee-jerks were active, there was no ankle clonus, and the plantar reflex gave a flexor response.

The boy was clean in his habits, played with his toy, said little pieces of poetry, and was contented.

His present condition (8 months later) has much improved. He can now walk, but is somewhat ataxic. There is less incoordination of his hands, but his articulation is still very defective.

The knee-jerks are present, there is no ankle clonus, but the plantar gives an extensor response.

No satisfactory view could be obtained of the discs.

(2) D. S. This child was shown at the Society one year ago. She is now 5 years old. She is the first and only child, and instruments were used at her birth. When last shown she pre-

sented a markedly incoordinate gait, incoordination of the arms, and difficulty in articulation. There was considerable difficulty in swallowing. The knee-jerks were active. She has improved a good deal in her walking but is still very ataxic.

(3) R. C., aged 6, was first seen in July, 1900. She was the sixth of seven children, all the others being healthy. The labour was prolonged, and was followed by a flooding. She began to walk when 4 years old, but tumbled about a good deal. She learnt to talk at 2 years, and has always been sharp, intelligent and clean in her habits. She is very slow in eating, and has difficulty in swallowing.

When first seen she walked in a very incoordinate manner, and was very liable to fall backwards. There is more incoordination in the right arm and leg than the left. There is no rigidity. The speech is slow and drawling, and not easy to understand. There is no nystagmus and the discs are normal.

The knee-jerks are active; there is no ankle clonus, and the plantar reflexes give flexor responses.

(4) L. T. This child was first seen in 1894, when 1½ years old, and she was in the Hospital for Sick Children under the care of Dr. Lees, with shaking of the limbs, inability to sit up, inability to talk, and nystagmus.

She was seen again in 1898, when 5 years old. She could then talk, but could not walk; there was much tremor of both hands and rotatory nystagmus.

The legs were rigid and ankle clonus was present. The intelligence was good, and the child was clean in her habits.

(II.) Dr. COLMAN.—*Case of (?) "mind blindness."*

Edwin C., aged 56. In September, 1900, had some kind of fit, after which he was "delirious" and did not recognise his surroundings. He recovered his ordinary state of health in a fortnight, but was found to be blind.

He is unable to distinguish objects well, there being evidently much dulling of ordinary light-perception; but he can count fingers and can make out the shape of objects that are shown him (*e.g.*, he says that a watch is round, has lines starting out from the centre and marks round its edge, but that he has no notion whatever what the object is. If he hears it or touches it he recognises it at once).

He will fail to recognise that an object is a book, but once he has assured himself by touch that it is a book, he will be able to pick out letters or even simple words, showing that he is not completely blind. He stumbles over obstacles, however, and has to be led about. His fundus is normal, and his pupils react quickly to light.

He has marked loss of memory for recent events, but there is little intellectual impairment beyond this.

In January he had another fit. There was no loss of consciousness. He was word-deaf for some hours, and had "gibberish aphasia," but next day there was no trace of aphasia.

(III.) Dr. WILFRED HARRIS.—*Case of syringomyelia following infantile paralysis.*

E. C., a married woman, aged 42, when aged 3 was stricken with infantile paralysis, damaging the left arm and leg and leaving, as permanent effects, left talipes equinovarus due to complete absence of the tibialis anticus and weakness of the extensor communis digitorum; in the left upper extremity some general smallness of the arm and forearm muscles with slight comparative weakness, and complete atrophy of the thenar eminence, and partial of the first dorsal interosseus, and general smallness of the bones of the hand. Beyond permanent chilliness of the left hand and some awkwardness in holding things, she suffered no disability until twelve months ago. For fourteen years she was in domestic service, and she afterwards married and had children. About March, 1900, she first noticed weakness in raising the left arm, and almost constantly since has complained of pain in the left shoulder and hand, and more recently in the spine and elbow. When first seen, last October, there was, in addition to the condition above described, marked weakness and some atrophy of the left deltoid, biceps, supinator longus supra and infra-spinati, and rhomboids, with fibrillary tremors in the deltoid and biceps. Partial tactile anæsthesia and analgesia above left elbow, extending over shoulder on to chest, back, and neck up to the mid-line and down to the xiphisternum. No scoliosis. Knee-jerks equal and brisk. Bilateral extensor plantar reflex. Jaw-jerk brisk. No bulbar symptoms nor nystagmus nor contraction of the visual fields. In January she complained much of pain in the cervical spine and elbow, but no spinal tenderness on pressure. She now volunteered that she could hold anything hot in the left hand without feeling it, though she is certain that the sensation of the hand was normal until recently. Since January the tactile anæsthesia and analgesia have invaded the whole of the left half of the body, head, limbs, and trunk, though the analgesia is most marked now on the left hand and wrist, and is also present on the right hand and lower part of forearm. The left knee-jerk is now much brisker than the right, and her left arm and leg have become much weaker lately. The electrical reactions show no reaction of degeneration in the deltoid, biceps, or supinator longus, but diminution of contraction and slight sluggishness to both forms of current.

(IV.) Dr. FARQUHAR BUZZARD.—*Atrophy of arm with anæsthesia over distribution of first dorsal root.*

A. O., widow, aged 40, under the care of Dr. Beevor, as an out-patient at the National Hospital, Queen Square.

Five years ago, whilst convalescent from Malta fever, the patient woke up one morning to find that she had lost power in her right hand, and that she had pain shooting down her right arm from the region of the neck and shoulder. The pain has recurred at intervals, and the loss of power has remained; three years later wasting of the hand muscles commenced, and has quickly progressed. At the present time, in an otherwise healthy woman, there is marked atrophy and palsy of the intrinsic thumb muscles of the right hand, some weakness of the interossei and of the long flexors of the fingers, complete analgesia and thermanæsthesia with impaired sensibility to touch over an area on the inner surface of the arm corresponding to the distribution of the first dorsal root.

(V.) Dr. RISIEN RUSSELL.—*Syringomyelia with "Charcot joint."*

J. C., male, aged 29 years. History of old trophic lesions and burn without pain on left hand. Weakness of left arm for three months, chiefly affecting abduction. Muscles unusually well developed, and none of them atrophied. "Charcot joint" left shoulder. Nystagmus on turning eyes to either side. Slight right facial weakness. Thermal anæsthesia left side of face, areas on trunk and both arms, analgesia and some tactile anæsthesia on left arm. Cold test tube causes feeling of burning left arm. Very slight lateral curvature of spine. Knee-jerks present. No ankle clonus.

(VI.) Dr. W. A. MARRIS.—*Cases resembling disseminated sclerosis.*

(1) J. B., male, aged 18, under the care of Dr. Ferrier. Family history is good. Patient was normal till three years

old, when he had scarlet fever. After this he was unable to walk and trembled all over. In three months he learnt to walk again, but the trembling is still present. The disease is not progressive. Patient's physical and mental development are very backward. The speech is thick and slow. The cranial nerves are normal, there is no nystagmus and no optic atrophy. The chief symptoms are the tremor of limbs and body, and slight spasticity in the legs. The tremor occurs on movement, disappears on resting, but it is not of crescendo type, and there is no ataxy in the arms. There are no sensory changes. The deep reflexes are increased, and the plantar reflexes are of extensor type. Patient has lately learnt to control his sphincters. Double pes cavus is well marked.

(2) H. W., male, aged 34, under the care of Sir W. Gowers. Patient's uncle had religious mania late in life, and a brother of the patient is suffering like the patient himself; otherwise the family history is good.

Patient was noticed to walk badly when twelve years old, and the illness has steadily progressed since. He has had no other illness. Mentally he is deficient, and his speech is markedly affected.

The optic discs are normal, there is no nystagmus, and the pupils react in every way. Slight facial weakness on the left side, and there is a very set expression of the face. The upper limbs show a fine tremor, but no definite ataxy or intention tremor. The legs are rigidly extended, and there is much spontaneous rectus-clonus. The plantar reflexes are extensor in type, the sphincters are unaffected. Pes cavus is present on both sides.

(3) C. W., brother of Case 2, aged 22, under the care of Sir William Gowers. Patient had pneumonia when 1 year old, and no illness since. Was later than the rest of the family in learning to walk and never strong on his legs. At 10 years of age difficulty in walking was distinct, and has slowly become worse. At present mental state is good, speech is slow and awkward. There is no pallor of the discs, no nystagmus, nor other affection of the cranial nerves. The upper limbs are normal, without any tremor or ataxy. The lower limbs are stiff, but all movements can be performed except dorsiflexion of the ankles. His gait is unsteady, and the feet drag. On the left side the toes alone reach the ground. There is slight patellar clonus, but no ankle clonus. The plantar reflexes are of extensor type. The sphincters are

unaffected. Pes cavus is marked on both sides, and there is considerable kyphosis in the dorsal region.

(VII.) Dr. Still.—*A case of paramyoclonus multiplex.*

Alfred R., aged 8½ years. His mother says he had "the jumps" in all his limbs for two years; the affection has varied in degree from time to time, but is worse the last few months.

No cause is assigned for its onset. He has had no previous illness, except measles and whooping-cough some years ago. No epileptiform attacks at any time.

Mother has had no miscarriages; patient is the first child, one other living and healthy. Father died with general paralysis three years ago.

Boy is fairly nourished; rather slow, but intelligent; speech deliberate and rather monotonous, with tendency to space words.

Sudden contractions of individual muscles, or portion of a muscle, not sufficient to cause movement of limbs, are seen, chiefly in the thighs where the quadriceps extensor of each side shows sometimes a single twitch, sometimes a short series of clonic contractions; the deltoid, triceps, biceps and scapular muscles are similarly affected, and, less often, the muscles of the leg, forearm and hand; similar twitchings are sometimes well marked in the rectus abdominis and the muscles of the back, and also in those of the nose and lips. The tongue also shows sudden irregular quiverings and twitchings, probably similar in nature to those elsewhere. A sudden sniffing sound, or slight grunt, which occurs occasionally, appears to be due to irregular contractions of respiratory muscles.

The affection appears to be chiefly symmetrical, but the movements on the two sides are not always isochronous. Voluntary movements seem to diminish the abnormal contractions, which are most noticeable when the child lies quietly in bed; they seem, however, to be absent in sleep.

Gait is normal, except that rarely the boy is pulled up suddenly by straightening of the knee owing to sudden contraction of the quadriceps femoris.

Electrical reactions are normal; sensation normal.

Tendon-jerks all very brisk; plantar reflexes normal (flexion).

BRAIN.

PART II., 1901.

Original Articles and Clinical Cases.

DESCENDING MESENCEPHALIC TRACTS IN CAT, MONKEY AND MAN; MONAKOW'S BUNDLE; THE DORSAL LONGITUDINAL BUNDLE; THE VENTRAL LONGITUDINAL BUNDLE; THE PONTO-SPINAL TRACTS, LATERAL AND VENTRAL; THE VESTIBULO-SPINAL TRACT; THE CENTRAL TEGMENTAL TRACT (CENTRALE HAUBENBAHN); DESCENDING FIBRES OF THE FILLET.

(*The tracts from the nuclei fastigii to Deiters's nuclei; descending thalamo-spinal fibres; a tract from the inferior collicular region to the ventral column of the spinal cord.*)

BY JAMES COLLIER, M.D., M.R.C.P.,
Pathologist to the National Hospital,
AND
FARQUHAR BUZZARD, M.B., M.R.C.P.,
Registrar to the National Hospital.

In the course of an experimental research upon the paths subserving certain reflex phenomena in the central nervous system of the cat and monkey carried out at the Laboratory of Pathological Chemistry, University College, London, under a license held by one of us (J.C.), we had occasion to make certain lesions of the mesencephalon.

The degenerations resulting from some of these experiments are here recorded. A series of cases in which destruction of the quadrigeminal and upper pontine region had occurred as a result of new growth or softening have been recently examined by us in the Pathological Laboratory of the National Hospital, and have given us the opportunity of comparing the experimental degenerations in the cat and monkey with those resulting from disease of the same region in man.

Our best thanks are due to Professor Vaughan Harley for kind permission to use his laboratory at University College, to Professor Victor Horsley for valuable criticisms and suggestions, and to the Medical Staff of the National Hospital for permission to make use of their cases. Dr. Newton Pitt most kindly sent us the case of mesencephalic abscess from Guy's Hospital. (This case will shortly be published by him in detail.)

METHODS.

The lesions of the mesencephalon were made by removing an area of bone over the posterior part of the sagittal suture, gently separating the hemispheres and puncturing through the corpus callosum with a cataract knife, without exposing the lamina quadrigemina. In every case the wounds healed by first intention. The animals were killed with an overdose of chloroform from fourteen to twenty-one days after operation. Both the experimental and pathological tissues were removed as soon as possible after death and were hardened in formalin for twenty-four to seventy-two hours and subsequently in Muller's fluid for eight days. For the Marchi method Bouche's sodium iodate modification was used, and for staining the medullated fibres Robertson's copper pyrogallic method and the Kulchitsky-Wolters method. The sections were cut in celloidin and mounted in serials.

The line diagrams were traced from the specimens by means of Edinger's apparatus, and the photographs were made with the same apparatus.

NOMENCLATURE.

Since some of the tracts dealt with in this communication have been described by authors under many different names, it will be convenient here to indicate these names and to add a brief anatomical description of each tract according to the most recent German investigators. We have adopted the terminology by which these tracts are referred to in recent French and German indexes.

Monakow's bundle.—Vierhügel-seitenstrangbahn; Abirrendes Seitenstrang-bündel (Monakow); Rubro-spinal tract (Pavlow); Le faisceau mésencephalo-spinale croisé laterale; Boyce's tract; Aussere Seitenstrang-bündel (Meynert); Fibres fondamentales du cordon lateral; Faisceau pré-pyramidal (Thomas, in the cord); Fasciculus tecto-spinalis cruciatus (Edinger); Lateral columnar tract (Boyce).

This tract was probably seen by Forel in 1884 in the mesencephalon and pons, but it was not traced to the spinal cord. It was first described by von Monakow in 1883, after hemisection of the cord below the pyramidal crossing in the dog, and subsequently after section of the lateral fillet at the level of the issuing fifth nerve. The development of the tract was investigated by von Bechterew and Held and more recently by Hösel; the fibres acquire their myelin early in the fifth month of fœtal life. The origin of the fibres in the red nucleus was ascertained by Held and Probst. Bechterew considers its place of origin to be the deep gray matter of the superior colliculus. The termination of the fibres among the cells of the ventral and dorsal horns of the spinal cord was shown by Probst and Rothmann.

This tract was investigated in birds by Münzner and Wiener and has been found constantly present in the rat, hedgehog, rabbit, guinea-pig, cat, dog and monkey. It was first described in man by Held.

The tract is composed of large fibres which arise in the cells of the red nucleus and issuing from its mesial aspect lie in a bundle immediately ventro-mesial to that structure. They cross the middle line sharply, decussating with those of the opposite side between the roots of the oculo-motorius in Forel's crossway—that part of the tegmental decussation lying between the fountain crossway of Meynert and the crossing of the brachium conjunctivum. The tract then turns caudalwards lying dorsal to the substantia nigra, ventro-mesial to the caudal end of the red nucleus and external to the issuing roots of the oculo-motorius. It then passes on to the dorsal aspect of the mesial fillet and gradually passing over this structure from within out, sinks through the angle of junction of the mesial and lateral fillet at the level of the motor nucleus of the fifth nerve and comes in relation with the mesial aspect of the spinal root of the fifth nerve. At the level of the exit of the seventh nerve the tract lies lateral to the superior olive, ventro-mesial to the spinal root of the fifth nerve and reaches the surface of the medulla crossed by fibres of the issuing seventh nerve. Below this it occupies the dorsal part of the fasciculus lateralis proprius of the medulla, lies immediately ventral to the spinal root of the fifth nerve and is mixed with the fibres of the lateral ponto-spinal tracts, the vestibulo-spinal tract and Gowers' bundle.

In the cervical cord the fibres are scattered over a wedge-shaped area with the base ventral and lateral, stretching from the mesial aspect of Gowers' bundle to the middle of the dorsal horn. The fibres which are traceable to the sacral region blend with the lateral pyramid below the cervical region and end among the cells of the ventral horn and in lesser extent also of the dorsal horns.

Contained also in Monakow's bundle are fibres which degenerate above points of section and are said to connect the ventral horn of the spinal cord with the nucleus ruber.

The dorsal or posterior longitudinal bundle needs no special description in this communication.

The ventral longitudinal bundle.—Vierhügelvorderstrangbahn ; Prædorsales Längs bündel (Tschermak) ; Faisceau *long. prédorsal ; Faisceau tecto-bulbaire* (*Pavlow*) ; *Faisceau marginale descendant* ; *Système du zone sulcomarginal* (*Marie*) ; *Fibres fundamentales des cordons antérieures* (*Hösel*).

This tract was first described by Held in 1892. The most important investigations and descriptions are those of Held, Tschermak, Pavlow, Boyce, Rothmann and Probst. Hösel has investigated the tract developmentally and has found that myelination of the fibres takes place in man during the fourth month of fœtal life shortly after that of the dorsal longitudinal bundle and before any other fibres than those of these two systems are medullated in the ponto-bulbar region.

These fibres have their origin in the deep gray matter of the superior colliculus close to the gray matter surrounding the aqueduct ; they course ventralwards and caudalwards to cross the middle line in Meynert's fountain crossway and then lie in a distinct bundle ventro-lateral to the dorsal longitudinal bundle, the distance between these two tracts increasing as they course down to the medulla where they come together and run into the anterior column of the cord. Collaterals leave the fibres of the ventral longitudinal bundle to end in the lateral stratum griseum centrale, in the lateral and contra-lateral oculo-motor nuclei and in the contra-lateral nucleus trochlearis and nucleus abducentis. The tract becomes much reduced in size below the nucleus abducens and can be traced in the anterior column of the cord as far as the lower lumbar region lying along the ventral border of the anterior fissure. The fibres terminate among the cells of the ventral horn some passing across the ventral commissure to end in the opposite ventral horn. The ventral longitudinal bundle consists entirely of fibres which degenerate downwards (Probst).

Held has described fibres running in the v. l. b. which, taking their origin from cells of the deep gray matter of the superior colliculus just behind the nucleus commissuræ dorsalis, run directly into the v. l. b. of the same side. (Ungekreutz Vierhügel-vorderstrangbahn).

The Ponto-spinal tracts.[1] — *Brücke-seitenstrangbahn Brückevorderstrangbahn; Fissurenstrang* (*Tschermak*); *Ungekreutzes Pyramidseitenstrang-system; Gekreuztes absteigendes Syst. aus dem mittl. und unteren Centralkern; Vorderseitenstrangreste der Mittelline* (*Held*).

These tracts were discovered and their origin and connections with the spinal cord partly described by von Bechterew using the myelination method in 1885. A good collective description of them is given by this author in the last edition of his treatise. Another excellent description is that of Tschermak. Fusari has described the tracts in man, degenerate after morbid lesions of the brain stem.

Separated as a crossed and an uncrossed system these fibres descend into the cord from the large cells of the reticular formation scattered from the medulla to the colliculus inferior, especially the nuclei centralis medius and lateralis medius of Bechterew and the central and lateral nuclei of Flechsig.

The mesial uncrossed system (Vorderseitenstrang-restes der Mittellinie of Held) runs into the dorsal longitudinal bundle, and can be traced as low as the conus teminalis occupying the borders of the ventral fissure (Fissurenstrang of Tschermak). They give many collaterals to the reticular formation of the medulla and others to end in the ventral horns of the spinal cord. (Uncrossed descending spinal nervous system from the reticular formation (Barker), Brucke-vorderstranghbahn of Bechterew.)

The crossed lateral system (Brucke-seitenstrangbahn of Bechterew) has its origin chiefly in cells of the nucleus centralis medius of Bechterew by fibres which immediately cross the middle line, and coursing through the d. l. b., turn caudally to lie between the d. l. b. and the inner limb of the facial; they then edge out into the olivo-restiform angle lying between the nucleus lateralis and spinal fifth, and run in the fasciculus proprius lateralis throughout the length of the spinal cord. In the cord the fibres lie somewhat scattered in the dorso-lateral part of the lateral pyramidal field, mesial to the fibres of Monakow's bundle and Gowers' bundle. They are said to terminate in the central region of the gray matter.

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[1] No name having been applied to these tracts in English, we venture to use this term as equivalent to the German Brucke-seitenstrangbahn and Brucke-vorderstrangbahn.

The vestibulo-spinal tract.—The descending tract from Deiters's nucleus; Absteigende Kleinhirnbahn (Biedl).

This tract consists of large fibres which have their origin in the cells of Deiters's nucleus, and also in the cells of the upper vestibular nucleus of von Bechterew. From these nuclei they take a curved course ventro-mesially, and crossing the facial knee sink to the mesial side of the facial nucleus and enter the fasciculus proprius lateralis medullaris. The fibres are mixed with those of Monakow's bundle, the ponto-spinal descending tracts, the antero-lateral spino-thalamic fibres and those of Gowers' bundle. The vestibulo-spinal fibres occupy however chiefly the mesial aspect of this region. In the lower medulla the tract lies between the nucleus lateralis and the remains of the anterior horn. In the spinal cord they lie in the anterior root zone and extend to the ventro-mesial angle of the ventral horn. They can be traced to the lower lumbar cord ending in the ventral horn of the same side.

This tract was first described by Held in 1890, and has been developmentally studied by Obersteiner. These authors gave the origin of the tract as above described. Marchi, Biedl, Basilewsky and others found this tract degenerate after hemi-extirpation of the cerebellum, and referred its origin to that organ. Russell showed that such degeneration subsequent to cerebellar hemi-extirpation was due to the coincident damage to Deiters's nucleus, and many writers have since confirmed this view.

The central tegmental tract.—Fasciculus tegmenti centralis (Barker); Centrale-Haubenbahn (von Bechterew) (Flechsig).

This tract was first described by von Bechterew, and subsequently by Flechsig. The former has recently investigated degeneration of this tract in man. It is one of the latest of all tracts to acquire myelin and for this reason is easily studied during development. It is a descending tract, but its origin, probably thalamic, has not been definitely ascertained. At the region of exit of the oculo-motorii it is fusiform in section and lies mesial to the nucleus ruber and outside the roots of the third nerve. At the level of the exit of the fifth nerve its position is quite central in each lateral half of the tegmentum; hence its name. The tract then drops ventralwards as it descends, and comes to lie between the inferior olive and the fasciculus lateralis proprius medullæ. Bechterew is alone in stating that it is continued below the olive as the olivenstrang (Dreikentenbahn of Helweg). "Abwärts scheint sich die Centrale Haubenbahn in der Olivenstrang des Ruckenmarks fortzusetzen." Other writers, however, deny the unity of the Centrale Haubenbahn with this triangular field.[1]

Descending fibres of the fillet.—Accessorische Schleife (*Bechterew*); *Lateral pontine bundle* (*Schlesinger*).

Very little that is definite can be found in neurological literature as regards centrifugal fibres in the mesial fillet. That certain of its fibres acquire their myelin late (after birth) suggesting centrifugal function has long been known through the researches of Flechsig and von Bechterew.

Hoche has described fibres in two cases in man which seem to have a definite course (the cases were thrombotic hemispheric lesions). The fibres left the capsule and occupied a position in the crus immediately lateral to the pyramidal fibres. In the pons they leave relation with the pyramidal fibres and coursing in a dorsal and mesial direction run caudalwards in the middle fillet whence some of the fibres pass to the nucleus facialis of the same and opposite side; other fibres enter the interolivary layer and course to the nuclei hypoglossi of the same and opposite side. Many other observers have noticed descending degeneration in the mesial fillet, but the course of the fibres has not been definitely traced.

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[1] In a recent publication Bechterew has, however, stated that the triangular tract arises on the surface of the inferior olive as the fasciculus peri-olivaris.

In both monkey and man lesions of the quadrigeminal region have produced atrophy of the fillet and inferior olive on the side of the lesion, but only after some time. It is difficult to distinguish such secondary atrophy from primary degeneration in pathological lesions when death occurs after the lapse of a considerable time.

EXPERIMENTAL DEGENERATIONS.

Experiment 1.—Cat. Lesion on either side of the middle line dividing the posterior half of the dorsal commissure, the fountain crossway and part of Forel's crossway; the habenular commissure was not divided.

Descending degeneration in both Monakow's bundles R. > L., both dorsal longitudinal bundles L. > R., the right ventral longitudinal bundle. (Plate I.—V.)

The lesion was produced in an attempt to divide the ventral part of the dorsal commissure, the fine cataract knife being introduced in the middle line with the cutting edge directed caudally, and the handle inclined strongly forwards. By an accidental movement of the knife a double linear lesion resulted, cutting a piece out of the posterior half of the dorsal commissure and running deeply on either side of the aqueduct along the mesial aspects of the caudal extremity of the red nuclei. On the left side the line of section extended to the interpeduncular space, on the right side to a point midway between the aqueduct and ventral surface of the mesencephalon lateral to the mid-line. The nuclei of the oculo-motorius and radix descendens trigemini were apparently not injured, each lesion lying external to the first and internal to the second, for there was no degeneration of the corresponding root fibres. The lesion did not involve the nuclei trochleares. The lesions were very sharply limited; there had been apparently little or no hæmorrhage.

In front of the lesion degenerate fibres were seen crossing in the lower part of the intact anterior half of the dorsal commissure to end in the nucleus commissurae dorsalis. On either side degenerate fibres ran forwards from the lesion to end in the gray matter along the sides of the third ventricle. The left fasciculus pedunculo-mamillaris was degenerate. The fasciculus retroflexus of Meynert was not degenerate. No degeneration was seen in the optic tracts, but a few fibres of the posterior commissure entered the external geniculate body on both sides.

Lateral to the lesions a few fibres passed outwards to end in the nucleus centralis superior. Ventral to the aqueduct no degenerate fibres were seen crossing the mid-line in front of the level of the exit of the anterior fibres of the oculomotorius.

From the cut ends of the dorsal commissure many degenerate fibres were seen coursing to the nucleus cent. sup. and nucleus com. dorsalis and many fibres to the dorsal longitudinal bundles on both sides. Fibres could be traced from the dorsal commissure to the ventral longitudinal bundle without any decussation below the aqueduct. Behind these, fibres from the nucleus lat. superior and deep gray matter of the superior colliculus crossed in Meynert's decussation and apparently passed into Monakow's bundle and the v.l.b. of the other side. No fibres were traced from dorsal commissure to the nucleus ruber.

A few of the anterior rootlets of the outgoing oculomotorius were degenerate, but only the first two sections showing these contained degenerate fibres.

In this region there was little or no degeneration in Meynert's fountain crossway, but in Forel's crossway, between the roots of the third nerve, were very many thick degenerate fibres forming apparently a complete decussation. These fibres commenced where the linear lesion skirted the mesial aspect of the caudal extremity of the nucleus ruber. The fibres crossing from right to left were not so plentiful as were those crossing in the opposite direction (the lesion did not extend so far ventral on the right side). Having crossed the roots of the third nerve, these fibres turned caudalwards and lay dorsal to the mesial end of substantia nigra, just outside the third roots, forming two conspicuous bundles—Monakow's bundles, the right containing twice as many fibres as the left, that is, in crossed relation to the more extensive lesion near the left red nucleus. In the region of the inferior colliculus these tracts passed on to the dorsal aspect of the mesial fillet and, edging outward in their course, came in relation with the mesial aspect of the spinal root of the fifth nerve, and passing to its ventral aspect maintained this position as low as the second cervical segment. At the level of the exit of the seventh nerve the tracts had a characteristic position, lying ventro-mesial to the spinal fifth, immediately external to the superior olive, and separated from the surface of the medulla by the fibres of the trapezium. In the lower medulla the fibres became somewhat separated and lay in the dorsal part of the fasciculus lateralis proprius, but some fibres came right down to lie immediately dorsal to the inferior olive. In the cervical cord the fibres occupied a fusiform area stretching from the mesial side of Gowers' bundle to the middle of the dorsal horn, and caudal of this region they were in the position of the lateral pyramidal tracts and were traced to the lowest sacral region.

Fibres leaving the tract in the medulla ran external to the spinal fifth and, passing through the restiform body, ended in the inferior vermis.

The termination of the fibres in the ventral and lateral horns was easily seen.

The dorsal longitudinal bundles contained many degenerate fibres, which occupied the dorsal and mesial parts of each bundle. These fibres were traced from the dorsal commissure. No fibres were traced across the fountain crossway (the lesions were outside the nucleus oculomotorius). The degenerate fibres were large and the left d.l.b. much more degenerate than the right. The tracts diminished in size considerably in the region of the sixth nucleus and in the cervical region. They were traced to the lower sacral region.

Comments.—The crossing and course of Monakow's bundles were typical. The position of the lesion mesial to the ventral end of the red nucleus more extensive on the left side, coupled with the greater degeneration of the right bundle, suggests the exit of the fibres from the ventral end of the red nucleus. The origin of some of the fibres appeared to be in the deep gray matter of the superior collicular region, an origin which Held and Boyce suggested.

The crossed relation of the degeneration of the d.l.b. and v.l.b. was striking.

Most of the fibres of the right v.l.b. seemed to come from the region of the posterior commissure, and since the tract was degenerated upon the right side only, it is possible that they had their origin in the gray matter close to that structure and correspond to Held's description of the non-crossing ventral colliculo-spinal tract. The passage of fibres from Monakow's bundle by the inferior peduncle to the cerebellum has not, so far as we are aware, been hitherto noticed.

It is worthy of note that notwithstanding the position of the lesion the "Centrale Haubenbahn" was not degenerated.

Experiment 2.—Cat. Sagittal linear lesion upon the right side, extending from caudal end of soft commissure to a point opposite the centre of the superior colliculus; it extended vertically from the ventral limit of the aqueduct to the interpeduncular space lying immediately mesial to the right red nucleus.

Degenerations below the lesion in the right dorsal longitudinal bundle, left ventral longitudinal bundle, and left Monakow's bundle. Fibres crossing in dorsal commissure to the ventral longitudinal bundle. (Plate VI.—VII.)

The lesion was produced in an attempt to cut the ventral fibres of the dorsal commissure, the knife introduced vertically in front of that commissure was tilted with the cutting edge back, so that the lesion produced was most extensive at its ventral limit, and viewed laterally was triangular in shape with the apex at the place of entry of the knife into the thalamus forward of the dorsal commissure on the left side and the base extending from the soft commissure into the region of Forel's decussation.

Some injury to the posterior part of the hemisphere on the left side caused degeneration of the left optic radiation running into the left superior colliculus, and slight degeneration of the left pyramidal tract.

In front of the lesion the fasciculus retroflexus of the right side showed some degeneration and a good many fibres ran forwards to end in the gray matter lining the third ventricle on the right side, a few crossing to the opposite side in Meynert's commissure.

From the outer side of the lesion many fibres crossed in the dorsal commissure and ran caudalwards along the lateral limit of the central gray matter on the left side, many fibres being traceable into the nucleus centralis superior, while fibres in a similar position ran caudalwards on the right side.

Those on the side for the most part ended in the nucleus oculo-motorius, nucleus trochlearis and nucleus fasc. long. dorsalis of the left side but some coursed ventro-mesially and ran throughout the brain stem as fibres of the left ventral longitudinal bundle. Those on the right side entered the posterior longitudinal bundle and were traceable as far as the sacral region of the spinal cord, occupying the most dorsal limit of the anterior column against the white commissure. From the inner side of the lesion in the neighbourhood of the right red nucleus coarse fibres crossed in Forel's decussation to occupy the position of Monakow's bundle on the left side, this tract being in every way similar to those described in the experiment 3. They were traceable as far as the lower cervical region, below which they were indistinguishable from the fibres of the pyramidal degeneration.

The fibres degenerate in the right posterior longitudinal bundle occupied its dorso-mesial aspect. The degenerate fibres of the left ventral longitudinal bundle had the typical course described on p. 181. They were very large fibres always somewhat scattered and, becoming much reduced in numbers in the region of the sixth nucleus and still further in the medulla, were traced to the lumbar region occupying the ventral border of the anterior median fissure.

The oculo-motor and trochlear nerve-roots, the left d.l.b., right v.l.b. and right Monakow's bundle showed no trace of degeneration.

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Comments.—The course of Monakow's bundle was typical and the absence of degeneration in the right bundle points to its fibres being entirely crossed.

The crossed relation of the degeneration of the d.l.b. and v.l.b. was again striking.

The degenerate fibres of the v.l.b. presumably came from the central gray matter close to the dorsal commissure as in the last case. This is in agreement with Held's results. The absence of degeneration in the "centrale Haubenbahn" is striking for it is difficult to conceive that it escaped injury on either side.

Experiment 3.—Rhoesus Monkey. Lesion in right superior colliculus and passing obliquely caudalwards, ventralwards and to the left, dividing Meynert's and in part Forel's decussation, damaging the nuclei oculo-motorii and trochleares and left red nucleus.

Degeneration below the lesion in both Monakow's bundles R. > L. reaching throughout spinal cord; right radix descendens trigemini, R. and L. dorsal longitudinal bundles and R. and L. ventral longitudinal bundles, and in both third and fourth nerves. (Plate VIII.—XII.)

The lesion was produced in the attempt to divide supposed fibres coming into the R. oculo-motor nucleus from the midthalamic region. A puncture, intended to be vertical with the blade of the knife transverse to the lines of the brain stem and a slight cut to the right, was made. The lesion was linear, transverse and localised. Roughly triangular in transverse section with apex above it extended from the centre of the R. sup. colliculus on its dorsal surface to the outer side of the left red nucleus crossing, the R. side of the central gray matter, touching the aqueduct at its ventral border, involving both third nuclei, both fourth nuclei, the left red nucleus and crossing Meynert's and Forel's decussations transversely, did not extend ventrally into the substantia nigra. The extent of the lesion in the direction of the length of the brain stem was very slight. The posterior commissure was not divided.

The section (fig. 1) through the anterior thalamic region shows no sign of lesion or of degeneration except in the internal capsule on the lateral side, due to accidental lesion of the hemisphere.

A section just below the thalamus (fig. 2), which reveals the fasciculus retroflexus in the greater part of its course, shows degeneration in the internal capsule of the lateral side, and also some degenerated fibres in the subthalamic white matter on the contralateral side running outwards horizontally in the position of the ansa lenticularis.

Sections passing through the posterior commissure and through the commissure habenulæ show no definite lesion, but the red nucleus on the contralateral side appears to be slightly damaged, and a few very fine fibres run from it in a dorso-lateral direction and quickly disappear.

In the posterior commissure a very few fibres are seen to be degenerated, some of them running out laterally into the white matter of the anterior corpus quadrigeminum on either side, others curving ventralwards in the direction of the median line and the red nucleus.

The degeneration in the pes pedunculi of the lateral side is still seen.

Degeneration is seen at the level of the superior colliculus in both tegmental decussations and in the oculo-motor nerve roots. In this region, too, there are a few degenerated fibres passing across the median line dorsal to the aqueduct.

Figure 3 represents a section through a lower part of the quadrigeminal region and through the decussation of the brachia conjunctiva. Degenerated fibres are seen in the descending root of the fifth nerve on the lateral side, in both dorsal longitudinal bundles, in both ventral longitudinal bundles and in a bundle of fibres henceforward to be called "Monakow's bundle," which is spreading out laterally on the dorsal surface of the fillet on either side. Comparing the two Monakow's bundles, one finds rather more degenerated fibres on the lateral side than on the contralateral side, due, probably, to the greater damage to the contralateral red nucleus.

At the decussation of the fourth nerve (fig. 4), Monakow's bundle is seen to have taken up its position in the bend of the lateral fillet and to lie just ventral to the lower end of the brachium conjunctivum.

This position is maintained in the upper part of the pons (fig. 5). A section taken at the level of the trigeminal nerve figures the degenerated descending root of the fifth, leaving its central position to reach the surface, passing through the bend of the fillet and leaving Monakow's bundle in the ventral angle which it forms with that structure (fig. 6). The dorsal and ventral longitudinal bundles are still quite distinct and the degenerated fibres in the pyramid of the lateral side are plentiful.

In the lower section (fig. 7), which is rather oblique and cuts the eighth nerve on the lateral and the seventh nerve on the contralateral side, Monakow's bundle is seen in its relation to both, and to the superior olive and spinal root of the fifth nerve. It lies just medial to the outgoing facial root, just ventral to the trapezium and is bounded on its internal surface by the superior olive. Below the facial nerve the external boundary is formed by the spinal trigeminal root (fig. 7). In the same section the dorsal and ventral longitudinal bundles are seen to be more diffuse and less well-defined.

In fig. 8 Monakow's bundle is seen on either side occupying a part of the olivary-restiform angle and is indistinguishable in its position from Gowers' tract at that level.

At the decussation of the fillet (fig. 9) the dorsal and ventral longitudinal bundles form a rather more compact mass; Monakow's bundle maintains its relation ventral to the spinal root of the fifth nerve.

The course taken by the dorsal and ventral longitudinal bundles in reaching the anterior columns of the cord is well seen in fig. 10 where some pyramidal decussation is taking place. In the same figure the relation of Monakow's bundle to the direct cerebellar tract is represented.

The result of the pyramidal decussation is to force Monakow's bundle further anteriorly and to destroy its immediate relationship with the spinal trigeminal root (fig. 11). All the degenerated fibres in the anterior columns now lie close alongside the median fissure.

Figures 12—14 represent sections of the cord taken in the cervical, mid-dorsal and lumbar regions respectively, and serve to show the relation of Monakow's bundle to the crossed pyramidal tract at the various levels of the spinal cord. It will be seen at once that it is only in the cervical region that the relative positions of these two tracts are at all easily distinguished owing to the characteristic shape of Monakow's bundle in this part. It seems to arise out of the middle of the crossed pyramid and to spread forwards in a sort of wedge, the apex of which reaches the surface of the cord in the anterior root zone region.

Monakow's bundle does not maintain this shape in the dorsal, lumbar and sacral regions of the cord, but is more incorporated with the crossed pyramid, appearing only as an anterior bulging of that tract.

In the sacral region (fig. 15) the number of fibres in the postero-lateral column of either side seems to be about equal, and these lie very near to the posterior root zone, a result which is probably due to the disappearance of most of the pyramidal fibres and the displacements dorsalwards of the fibres of Monakow's bundle.

Comments.—The degeneration in this experiment demonstrates the crossing of the fibres of Monakow's bundle and of the ventral longitudinal bundle in the tegmental decussations.

The greater degeneration in the Monakow's bundle on the side crossed to the lesion of the red nucleus suggests that these tracts are entirely crossed. The relation of these bundles to the radix descendens trigemini is well shown.

The fibres from the region of the dorsal commissure to the ventral longitudinal bundle of the same side again support Held's argument of uncrossed colliculo-spinal fibres.

Experiment 4.—Rhoesus monkey. Lesion in a sagittal direction to the right of the middle line extending from the soft commissure to the upper limit of the inferior colliculus.

The dorsal and habenular commissures were divided and the fasciculus retroflexus of the right side was involved.

Descending degeneration in the fillet as far as the medulla on both sides, in both descending trigeminal roots, in both dorsal longitudinal bundles and both ventral longitudinal bundles. (Plate XIII.—XIV.)

The lesion was produced in an attempt to divide the posterior commissure. The knife had entered obliquely at the middle of the soft commissure to the right of the middle line, and a linear lesion resulted which divided structures dorsal to the aqueduct on the right side and crossing the aqueduct from right to left had divided structures ventral to the aqueduct on the left side. The right fasc. retroflexus was injured, the dorsal and habenular commissures were completely severed to the right of the middle line. The line of the lesion entered the aqueduct and reached the third nuclei; from this point some damage, probably from hæmorrhage, extended ventrally and to the right into the nucleus lateralis superior and deep gray of the superior colliculus. The fourth nuclei were injured on both sides, but the lesion did not extend so far ventrally as the dorsal longitudinal bundles. Neither Meynert's nor Forel's crossways were damaged. The last trace of the lesion was seen in sections still showing the fountain crossway in the position of the right fourth nucleus.

Figure 1 is drawn from a section taken through the middle commissure showing the anterior end of the lesion. The only degenerated fibres at this level are those in the ansa lenticularis of the lateral and those in the internal capsule of the contralateral side.

The fasciculus retroflexus of the lateral side has been badly damaged by the lesion and many degenerated fibres are seen running towards its caudal extremity (fig. 2).

Figure 3 shows the destruction of the posterior and habenular commissures. From either side of the posterior commissure spring degenerated fibres, some of which run out laterally in the direction of the upper fillet, whilst others take a curved course ventralwards and either decussate in the mid-line or end in the neighbourhood of the red nucleus. Those fibres which run into the upper fillet are much more plentiful on the lateral than on the contralateral side.

An extension outwards of the lesion on the lateral side of the Sylvian aqueduct in the superior quadrigeminal region increases the number of degenerated fibres coming from that side and entering into the formation of Meynert's decussation (fig. 4). At this level degeneration is seen in both dorsal longitudinal bundles, but more marked in that of the lateral side. The fibres of the fillet are seen sweeping round in a ventral direction; numerous on the lateral, scanty on the contralateral side. Both third nerves and the contralateral pyramid are degenerated.

Figure 5 represents a section taken through the decussation of the brachia conjunctiva. Here it will be seen that the fibres of Meynert's decussation have now turned caudalwards and lie just ventral to the dorsal longitudinal bundles. These are the fibres of the ventral longitudinal bundles and rapidly take a more ventral position, running, as they do so, amongst the decussating fibres of the superior cerebellar peduncles. At this level degeneration of both descending trigeminal roots is noticed and from each a few fibres appear to spring and run out in a ventro-lateral direction. The degenerated fillet fibres do not take identical positions on either side, those on the lateral side turning caudalwards earlier and at a greater distance from the mid-line than those on the contralateral side. The lowest end of the lesion is seen in the same figure in the shape of slight damage to the ventral wall of the aqueduct.

At the decussation of the fourth nerves (fig. 6) the ventral longitudinal bundles have taken up the position which they maintain to the lower end of the pons.

Figure 7 represents the degenerated descending trigeminal roots making their way to the surface to join the main nerve-trunk.

In the medulla (fig. 8) the ventral and dorsal longitudinal bundles have become more or less fused. The fillet fibres on either side have become scattered and no longer constitute a compact bundle; some are seen in the reticular formation and some hug the dorsal and lateral aspect of the inferior olives. On the contralateral side there are a few degenerate fibres belonging to the pyramidal system which lie lateral to the main pyramidal mass, and these fibres can be traced into the cervical cord in an uncrossed path (see figs. 9 and 10).

At the level of the decussation of the fillets the fibres belonging to the longitudinal bundles sink further ventralwards and are forced into a more lateral position by the decussation. Tracing these fibres into the cervical cord (fig. 10), it will be noticed that on the contralateral side more fibres remain near the lip of the anterior median fissure; these may be supposed to belong to the ventral longitudinal bundle system, whilst the fibres which have sunk deeply along the margin, being more numerous on the lateral side, are probably continuous with the dorsal longitudinal bundles.

Fibres in both these positions can be traced in decreasing numbers down to the lumbo-sacral region of the cord.

Below the decussation of the pyramid a few degenerated fibres are to be found in the postero-lateral region of the contralateral side, and these undoubtedly are uncrossed fibres from the degenerate pyramid above.

In figs. 10 and 11, taken respectively from sections in the cervical and sacral parts of the cord, degenerated fibres belonging to a root system appear in the posterior columns. For some unknown reason, degeneration of one or more posterior roots at the lower extremity of the cord has taken place, and the fibres can be traced through their entire course upwards into the posterior column nucleus of the lateral side.

Comments.—The degeneration of the fillets seemed to correspond with the lateral expansion of the lesion in this animal as compared with the other experiments. As regards the ventral longitudinal bundles, the same remarks apply in this as in the other cases.

Experiment 5.—Cat. Lesion of right optic thalamus limited between soft commissure and dorsal commissure; not reaching wall of ventricle and not destroying corpus subthalamicum.

Degeneration behind lesion in post commissure, and in a bundle lying immediately ventro-lateral to the dorsal longitudinal bundle of side of lesion and extending into the medulla and spinal cord, in stratum opticum and in upper fillet. (Plate XV.)

The lesion was produced in an attempt to divide the frenulum habenulæ of the right side by puncture through the corpus callosum. There was a somewhat rounded hæmorrhage measuring 5 mm. transversely and 3 mm. vertically (the puncture mark in the surface of the thalamus was midway between the soft and dorsal commissures); it involved chiefly the anterior part of the red nucleus and region immediately external to this. From a slight injury to the left marginal convolution some fibres of the left pyramidal system were degenerate. In front of the lesions some degenerate fibres ran outwards, forwards and downwards, and ended in the right corpus Luysii, others running into the right ansa lenticularis; many ran outwards and into the right optic radiation. The optic tracts, Vicq D'Azyr's bundle and Meynert's bundle and commissure, with the ganglion habenulæ, were normal.

From the mesial aspect of the lesion a few fibres crossed the dorsal commissure and immediately disappeared in the neighbouring gray matter.

From the caudal limit of the lesion a copious stream of degenerated fibres ran back, and in sections still showing the dorsal commissure were seen to divide into three groups, "A" entering the stratum opticum of the superior colliculus, "B" entering the region of the fillet, "C" forming a compact bundle at some distance ventro-lateral to the Sylvian aqueduct.

Group "A," composed of very coarse fibres, gradually broke up and disappeared in the stratum opticum. Group "B," composed of numerous very fine fibres, lost many fibres in the region of the red nucleus and substantia nigra, and entered the right mesial fillet. Dropping ventrally in their course and gradually thinning out, these fibres were no longer traceable at the level of the exit of the trigeminus.

Group "C" formed a most conspicuous and compact bundle of large fibres which coursed caudalwards, maintaining a constant position ventro-lateral to the right dorsal longitudinal bundle. (There were no degenerate fibres in either d.l.b.) This tract was everywhere quite separate from the d.l.b. and from the ventral longitudinal bundle.

The fibres became much reduced in number but not in size below the level of sixth nucleus. In the lower medulla the fibres lay on either side of the middle line, having a position between those of the d.l.b. and the v.l.b. and lateral to both. They entered the anterior column of the cord with these tracts, reduced in number below the decussation of the pyramid to about six coarse fibres, some of which were traced to the lumbar region of the cord. With the exception of the slight degeneration of the left pyramid, which was traceable to the sacral region, no other degeneration was found in the nervous system.

Comments.—The degeneration of the fillet fibres very intense at first and rapidly diminishing caudally, none being traceable into the medulla, was striking. They apparently ended in the nuclei pontis. We have not been able to find any mention of such fibres in the literature.

The fibres of group "C" were certainly thalamic in origin and held a straight course. They were certainly not fibres of the Haubenbahn. Nor from their course and termination did they appear to belong to the group of the mesial fillet.

In sections of the pons they at first sight appeared to be part of the ventral longitudinal bundle. They did not arise in the gray matter of the superior colliculus and do not therefore correspond with Held's uncrossed tecto-spinal fibres. The tract was apparently an uncrossed descending thalamic tract, to the ventral columns of the cord.

PATHOLOGICAL DEGENERATIONS.

Case 1.—Tumour involving almost the whole of the dorsal two-thirds of the mesencephalon in the region of the superior colliculus involving the pulvinar thalami and the left capsule.

Degeneration below the lesion in the left pyramid; both central tegmental tracts, both Monakow's bundles, both brachia conjunctiva R. > L.; both descending roots of the fifth nerve and in both mesial fillets L. > R. (Plate XVI.—XVII.)

Clinical abstract.—B.M., aged 15 years, admitted into the National Hospital on July 6, 1900, under the care of Sir William Gowers.

Total duration of symptoms one year.

Chief symptoms: Right hemiparesis, total hemianæsthesia to all forms on right side; right knee-jerk absent, complete incontinence of sphincters, ocular palsies. Blindness from consecutive optic atrophy.

Anatomy.—The tumour was strictly limited and hard in consistence with many recent hæmorrhages in its substance. It was a granuloma perhaps of tuberculous nature. It involved the left thalamus and left internal capsule, slightly the right thalamus. Its upper limit in the thalamus was the soft commissure. A tongue of growth extended into the left tegmental region as low as the lower part of the inferior colliculus. A section at the level of the anterior part of the superior colliculus showed that the growth involved the whole mesencephalon at that level, except the right pyramid and substantia nigra and a portion of the ventro-lateral region on the left side and the commencement of the crossing fibres of the pons. In the first section below the main lesion clear of the "traumatic degeneration" the left pyramid was intensely degenerated and the degenerate fibres showed signs of having been more recently affected than those of the other tracts about to be described. The pyramidal degeneration as regards its course, decussation and termination showed no peculiarity. Both superior cerebellar peduncles showed a great many degenerate fibres R. > L. which were traced to the interior of the dentate nucleus and to the cortex of the lateral cerebellar lobes. Both fourth nerves and both descending roots of the fifth nerve were degenerate and showed no peculiarity worthy of special mention. The dorsal longitudinal bundles and ventral longitudinal bundles were much degenerated and of the former chiefly its dorsal and mesial fibres. The v.l.b.'s were conspicuously distinct with their passage into the anterior columns of the cord where they mixed with the d.l.b.'s and anterior pyramidal degeneration. They were situated some little distance lateral to the raphe and sank farther and farther from the d.l.b.'s in their course caudalwards. The fibres were easily distinguishable from the fibres of the degenerate fillets in the inter-olivary layer by their being much larger. Both

Monakow's bundles were degenerated and their course so closely resembled that already described in the cat and monkey that it is sufficient here to describe certain important points only. The decussation of these fibres in Forel's decussation was plainly seen. From the exit of the facial nerve to the second cervical segment these tracts kept much more closely to the ventro-mesial aspect of the spinal fifth and did not reach the surface in the olivo-restiform angle nor spread downwards to the inferior olive. In the region of the decussation of the pyramids they were situated far more dorsally and deeper than in the cat and monkey and were ventral to the substantia gelatinosa. When the pyramid had crossed the tracts had the form of an elongated equilateral triangle with narrow base on the surface of the cord and apex touching the outer surface of the lateral pyramidal tract. Below this the tracts became fusiform and the external point of the spindle turned forwards to the ventral root region. The fibres were not distinguished from the crossed and contra-lateral pyramidal degeneration below the fifth cervical segment.

The fibres were large but few in number. It is possible that on account of the age of the degeneration much of the Marchi reaction had been lost. (Indeed specimens of this case mounted in the ordinary way showed little or no trace of any degeneration except the pyramidal an hour after they were mounted, but only a dark cloud. This was obviated by rapid clearing, mounting uncovered on a glass plate in hot thick balsam and drying rapidly in hot air. Hamilton's modification of Heller's method for fixing old degeneration, combined with rapid desiccation of the balsam was also used.)

The left mesial fillet showed many degenerate fibres, a few only being present in the right mesial fillet. The former descended into the left half of the interolivary layer, where many entered the stalk of the left inf. olive, and some crossing the raphe entered the right inf. olive. Many of these fibres passed downwards and disappeared in the region of the pyramidal decussation. Some of them crossed in the fillet decussation, and were traceable into the region of the dorsal column nuclei. None were traced into either lateral column of the cord. The degenerated fibres in the right fillet had a similar course. Next to that of the left pyramid the most conspicuous degeneration was that of the " Centrale Haubenbahn " on either side. At the level of the posterior part of the decussating brachii two large fusiform areas of degeneration were visible, plainly showing to the naked eye in the degenerate decussation the long axis, vertical and reaching on either side from the d.l.b. almost to the mesial fillet. Traced upwards into the region of the lesion the right tract was not obscured by the lesion for some distance and lay immediately mesial to the hinder end of the red nucleus and above a conspicuous degenerated Monakow's bundle.

Traced downwards the tracts kept close and mesial to the uprising brachia, and at the level of the fourth decussation formed the " star " to the crescentic outline of each brachium; the central position of the tracts in each lateral half of the dorsal brain stem which led Bechterew to name the tract " Centrale Haubenbahn " was conspicuous. Immediately below this level each tract sank sharply down and ran as a large round bundle close dorsal to the middle of the mesial fillet, and where the latter structure began to break up in the interolivary layer it crossed ventro-laterally and lay first close dorsal and then external to the inferior olive. The fibres could be traced round the sheath of the olive and between this and the pyramid, much reduced in numbers, and appeared to end in the olive.

Comments.—This case is interesting from the association of complete hemianæsthesia to all forms with a lesion completely destroying one half of the tegmental region. The important points in connection with the degenerations below the lesion are:—(1) The presence of Monakow's bundles, their decussation in Forel's crossway, and their course and terminations being identical with the same tracts described in the lower animals; (2) The presence of the central tegmental tract in man and its termination in great part in the inferior olive; (3) The separation of the ventral longitudinal bundles as distinct and well marked tracts; (4) The descending degeneration in the mesial fillets and in the brachia conjunctiva.

Case 2.—Abscess of the tegmental region, anterior limit at the foramen of Monro. Posteriorly reaching into the superior medullary velum as far as the commencement of the vermis. Involving chiefly the left corpus subthalamicum and left quadrigeminal region. At the level of the upper part of the pons the whole of the mesencephalon dorsal of the mesial fillet was destroyed.

Degeneration in a tract on either side, running from the superior medullary velum to Deiters's nucleus.

Degeneration of both dorsal longitudinal bundles, both ventral longitudinal bundles, both central tegmental tracts, both brachia conjunctiva, both descending trigeminal roots, both Monakow's bundles, and in both mesial fillets, and (slight) in both pyramids.

Marked atrophy of the left inferior olive. (Plate XVIII.—XIX.)

Below the thalamic region the abscess cavity was confined entirely to the tegmental region, and posteriorly spread into the superior medullary velum, pressing the superior cerebellar peduncle ventralwards. Its posterior limit was on the left side in the roof of the fourth ventricle.

The pyramidal degeneration was slight and was more marked upon the left side. The central tegmental tracts were first seen ventral to the lesion and mesial to each distorted superior brachium. They descended to the dorsal aspect of each mesial fillet, and in this position each had the form of a large round bundle. They then dropped ventralwards at the commencement of the interolivary layer to course round the external and then the ventral aspect of the inferior olive, and were lost in the interolivary layer immediately dorsal to the pyramids. The fibres of these tracts were very fine. No connection of these tracts with the triangular field could be made out.

The degenerated fibres in the mesial fillet descended, after giving off many fibres to the region of the facial nucleus, into the interolivary layer, and many fibres entered the inferior olive; other fibres were traced across the fillet decussation to the posterior column nuclei.

The course of the degenerated fibres of Monakow's bundle was typical. In sections taken rather obliquely through the pons, the dorsal limit of the section being some distance in front of the ventral limit, the central tegmental tract and Monakow's bundle were seen on each side, their fibres running in the plane of the section parallel to one another as they descended to the olivo-restiform angle, the two tracts enclosing the superior olive and nucleus facialis. The contrast of the coarse fibres of Monakow's bundle and the fine fibres of the central tegmental tracts was very striking.

In the fasciculus proprius lateralis medullæ these two tracts occupied the ventral limit and the dorsal limit of this region respectively, the central tegmental tract lying in contact with the olive, and Monakow's bundle being situated immediately ventral to the spinal root of the fifth.

In the cord Monakow's bundle took the form of a fusiform area, stretching in a dorso-mesial direction deeply into the lateral column from the lateral limit of the anterior roots. Below the upper dorsal region the fibres were blended with the few degenerate fibres of the lateral pyramidal tract.

The dorsal and ventral longitudinal bundles were distinct as far as the fillet decussation, and were followed to the sacral region lying on either side of the ventral fissure.

Traceable directly out of the posterior part of the lesion in the superior medullary velum, a well-marked band of fibres ran ventrally and caudally along the mesial aspect of each superior cerebellar peduncle, lying between this and the ependyma. These tracts were traceable directly into Deiters's nucleus where they broke up and ended. None of these fibres could be traced into the region of the nucleus of the vagus.

Comments.—This case serves to demonstrate Monakow's bundle and the central tegmental tracts as tracts which degenerate downwards in man, in lesions of the mesencephalon. The localised lesion of the valve of Vieussens, with a tract of degeneration on either side running thence to Deiters's nucleus, is of interest. These tracts are very conspicuous in transverse sections of the pons of infants stained by Pal's method. They have been studied developmentally by Kolliker and by Edinger, using degeneration methods, in both man and the higher vertebrates. They are described as having their origin in the nucleus fastigii and possibly also in the nucleus emboliformus and nucleus globosus, and as running on one hand to the nucleus of Deiters, on the other hand to the nucleus vagi. Kolliker's investigations by Golgi's method suggest, however, that some of the fibres of these tracts have their origin in the nucleus vagi and in Deiters's nucleus and ascend to the roof nuclei of the cerebellum.

Case 3.—Tumour of the pons extending from the region of the inferior colliculi to the junction of the pons and medulla, more extensive upon the left side.

Descending degenerations in both pyramids L. > R.; in the left mesial fillet and central tegmental tract; left fasciculus proprius lateralis of the medulla; in both dorsal and the right ventral longitudinal bundle; left spinal trigeminal root. Atrophy of the left inferior olive. (Plate XX.)

Clinical abstract.—R. D., aged 42 years, admitted into the National Hospital, under the care of Dr. Ormerod, on March 19th, 1901. Duration of symptoms ten months. Course of symptoms. Numbness of right hand and leg, right hemiplegia; numbness of left hand, paresis of left hand; complete bone deafness of left ear; left fifth, sixth and seventh nerve palsies; dysphagia and dysarthria. (The left leg had been amputated.)

The tumour was a glioma which had undergone degeneration and it contained many hæmorrhages. It implicated almost the whole of the left half of the pons from the inferior colliculus to the upper end of the inferior olive, and it extended across the middle line in the middle pontine region.

The degeneration of the left pyramid presented no peculiarity. The degeneration in the right pyramid was entirely confined to the outer third of the pyramid in the medulla; these degenerate fibres crossed entirely at the decussation and occupied a position in the lateral column close to the dorsal horn. The spinal fifth was traced to the upper part of the third cervical segment. The central tegmental tract was first recognisable lying above the mesial fillet and had a characteristic course round the olive. It was traced between the pyramid and olive, losing many fibres on the latter, and some of its fibres seemed to end by entering the hilum of the inferior olive, and possibly some crossed the middle line to enter the contralateral inferior olive.

The degenerated fibres of the fillet in part entered the inferior olive, but most of them crossed in the fillet decussation and ended in the region of the posterior column nuclei. The degeneration of the right ventral longitudinal bundle was distinguishable from that of the dorsal longitudinal bundle as far as the decussation of the pyramids and the degeneration of these tracts was traced to the lowest sacral region lying on either side of the ventral fissure. The degeneration of the fasciculus proprius lateralis medulla occupied the dorsal part of the olivo-restiform angle, ventral to the degenerate spinal root of the fifth. In the spinal cord this degeneration was arranged as a band of scattered fibres stretching from the anterior column degeneration to the outer limit of the anterior roots, and continuous with this a copious wedge-shaped area of degeneration with base at the surface of the cord dorsal to the anterior root, and apex stretching into the crossed pyramidal tract. This degeneration in the antero-lateral region was traced in this position to the lumbar region.

Comments.—The degeneration in the fasciculus proprius lateralis medullæ was probably made up of Monakow's bundle, the lateral ponto-spinal tracts, and the vestibulo-spinal tract. The distribution of these fibres on reaching the spinal cord was that of the vestibulo-spinal tract (Russell) and Monakow's bundle. The termination of the central tegmental in the triangular area of Helweg was most certainly not present.

Case 4.—Tumour of the pons and upper medulla with practically complete destruction of the lower pons; the left pyramid and the left inferior olive being the only recognisable structures in sections stained by Heller's method.

Degeneration in both ventral columns, both antero-lateral columns, both lateral columns; the direct cerebellar tract and Gowers' tract alone escaping. (Plate XXI.)

Clinical abstract.—M. S., aged 5½ years, admitted into the National Hospital, under Dr. Ferrier, on September 7, 1900. Duration of symptoms nineteen months. Course of symptoms: Diplopia, right hemiplegia and hemiataxy; paralysis of the left fifth nerve; incontinence of urine; optic atrophy; complete flaccid paraplegia; loss of deep reflexes.

The growth was a hard glio-sarcoma. It extended from the level of the exit of the fifth nerve to the middle of the inferior olives. Its sagittal extent was much greater upon the left side. At the level of the junction of the pons and medulla it involved almost the whole transverse section of the brain stem.

In the cervical region of the spinal cord almost the whole of the anterior and lateral columns were degenerate, with the exception of the regions occupied by the direct cerebellar and Gowers' tracts, some fibres of the anterior column around the median fissure, and some fibres round the gray matter of the ventral horns. The regions of the pyramidal tracts and antero-lateral descending tracts showed much old degeneration by the Marchi method, and the degeneration was still more striking in sections stained by Weigert's method and van Gieson's method. There was some degeneration in all the anterior roots of the cervical region.

In the other regions of the spinal cord the degeneration occupied the same positions, but became less extensive in lower regions of the cord; it was traced to the lowest sacral region. In the sacral and lumbar regions there was marked degeneration of the posterior roots (not figured).

Comments.—The maintenance of life, even of a merely vegetative nature, was remarkable, considering the extensive nature of the lesion, which apparently amounted to a total transection of the brain stem at the junction of pons and medulla. The degeneration was very great in the ventral and lateral columns, and practically only in the ascending tracts and in the region of the short internuncial fibres round the gray matter, were normal myelin sheaths to be seen, and consecutive sclerosis had altered the shape of the spinal cord; the ventral regions being much thinned. The degeneration of the anterior roots in the cervical region was interesting. It does not seem probable that it was of the nature of a degeneration from disuse, for we have failed to find any such degeneration below the lesion in cases of total transverse lesions of the spinal cord, even when life has been prolonged for many months after such injury. It is possible that this degeneration of the ventral roots is of the same nature as the degeneration of the posterior roots, which frequently occurs in connection with intracranial tumours. Both the intramedullary and extramedullary roots were degenerated. There was in this case much recent degeneration of the posterior roots, and this was most marked in the sacral region. This has not been inserted in the diagram.

Case 5.—Tumour of left centrum ovale, involving the left internal capsule. A localised patch of recent softening was present in the superior collicular region, involving the nucleus lateralis superior, the upper part of the substantia nigra, the upper fillet, and, slightly, the outer side of the red nucleus on the left side. A small hæmorrhage was present in the posterior inferior part of the left inferior colliculus.

Degeneration below the lesions in the left pyramidal tract, accessory fillet, and mesial fillet.

From the lesion in the inferior colliculus a large fibred tract descended, having a dorso-lateral situation in the pons and medulla, and in part, joining the pyramid at the decussation, appeared to run as a ventro-lateral tract in the cord.

G. B., aged 54 years, was admitted into the National Hospital under the care of Sir William Gowers. Duration of symptoms eight weeks. No symptoms present till eight weeks before death, when a sudden attack of right hemiplegia and aphasia occurred. A second attack ten days later. No optic neuritis, headache, nor vomiting. No hemianæsthesia. Deep reflex increased. (Plate XXII.—XXIII.)

The tumour was a large soft glioma with much softening and hæmorrhage in its substance. It involved the whole thickness of the central part of the internal capsule, but the anterior part of the anterior limb and the posterior part of the posterior limb escaped. A recent softening was present in the left mid-brain at the level of the outgoing third nerve, involving the region of the upper fillet, the outer part of the nucleus lateralis superior and nucleus ruber, and the upper part of the substantia nigra. A recent hæmorrhage was present, involving the left lateral fillet in the angle between the left superior brachium and left mesial fillet.

The degeneration of the left pyramidal fibres occupied the middle two-thirds of the crus. At the decussation of the pyramid well-marked ventral and lateral homolateral bundles were present, and a ventral crossed bundle was present.

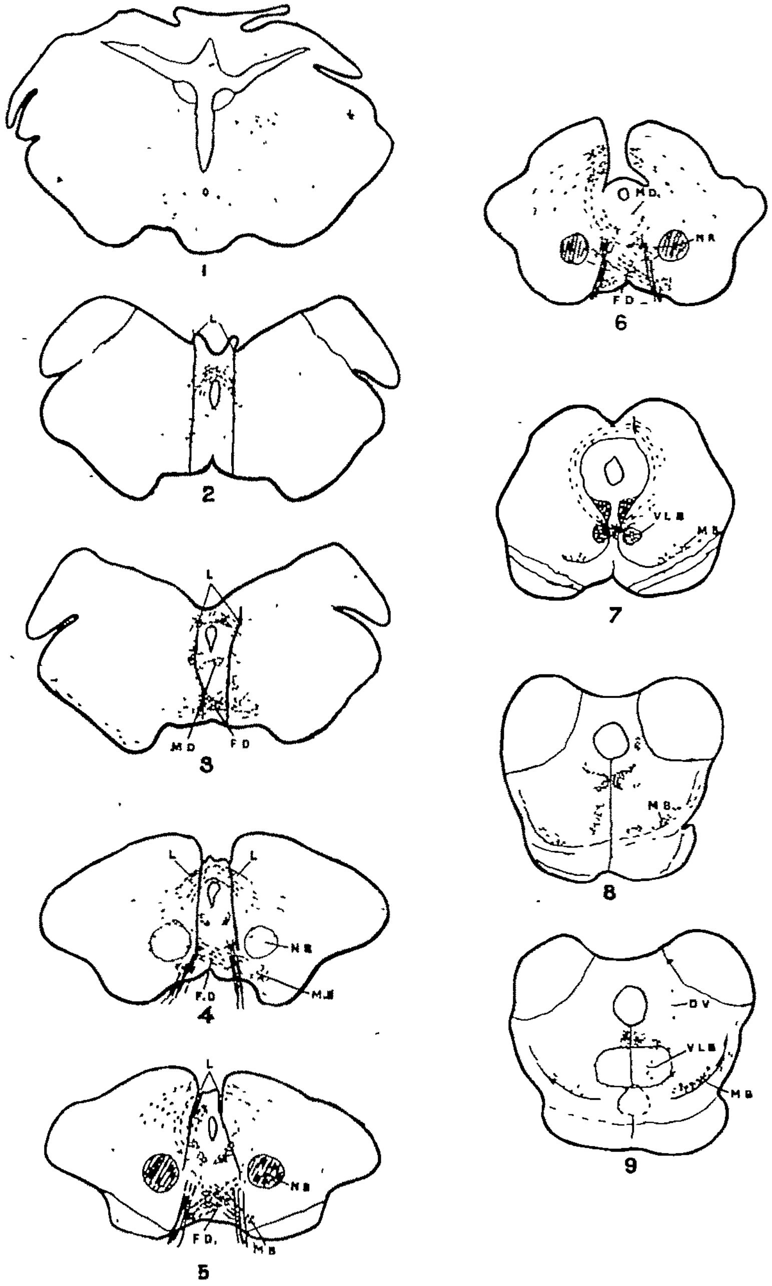
Conspicuous in sections of the upper part of the mid-brain was a large bundle of fibres lying in the concavity of the left crus, between this and the substantia nigra (accessory fillet). This bundle rose above the most lateral fibres of the crus in its descent and on entering the pons left relation with the pyramidal fibres and entered the mesial fillet, some of the fibres lying in layers between this structure and the pyramidal bundles. In the pons many of these fibres were seen coursing ventrally, apparently to enter the pyramidal bundles, others ended in the nuclei faciales and hypoglossi of the same and of the opposite side, others descended into the interolivary layer and apparently entered the left pyramid.

Distinguishable from the accessory fillet fibres were other degenerate fibres in the mesial fillet traceable from the lesion in the quadrigeminal region. These were followed into the interolivary layer of the left side.

From the lesion in the region of the left lateral fillet a well-marked band of large fibres was traced downwards, having a straight course through the lateral region of the pons in the upper part of its course lateral to the superior brachium below lateral to the restiform body. It reached the surface of the medulla at the level of the exit of the tenth nerve where it lay on the dorso-lateral surface of the restiform body as a very compact triangular tract with the base on the surface. In the lower medulla these fibres ran sharply downwards over the surface of the lateral column to the antero-lateral region.

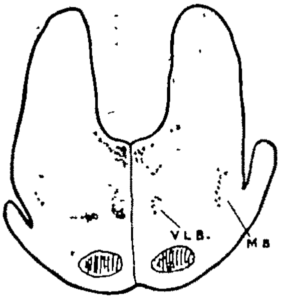
Comments.—This case exemplifies very well the four tracts into which the pyramid splits at the decussation

PLATE I.



Cat. Division of dorsal commissure, Meynert's decussation and Forel's decussation in part. Degeneration below the lesion in both Monakow's bundles, R. > L.; both dorsal longitudinal bundles, and in the right ventral longitudinal bundle. *Projection drawings.*

PLATE II.



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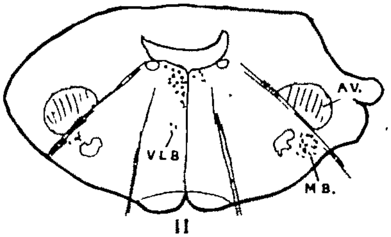

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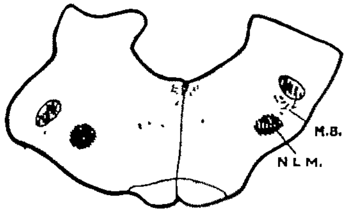

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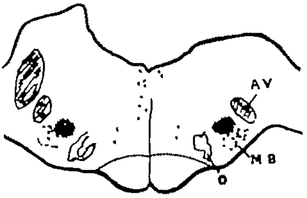

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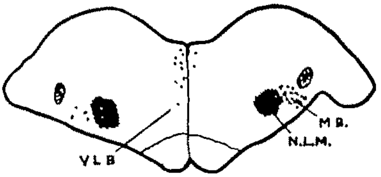

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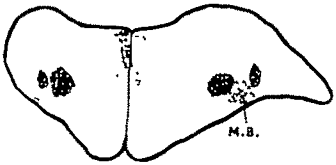

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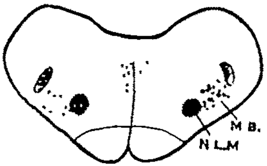

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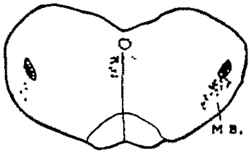

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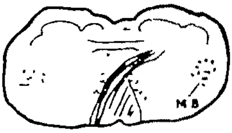

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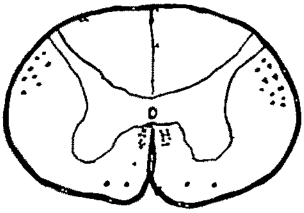
PLATE III

19



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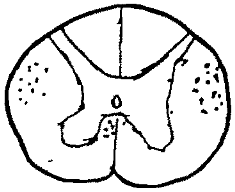

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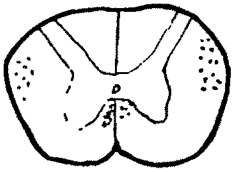

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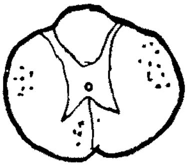

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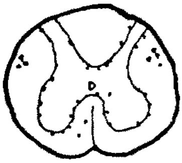

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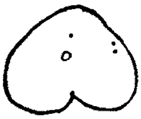

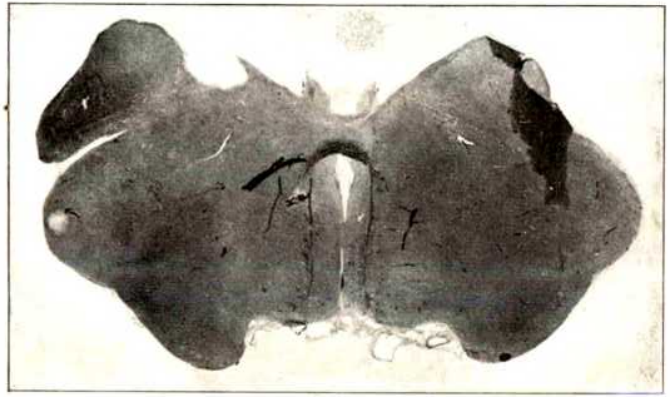

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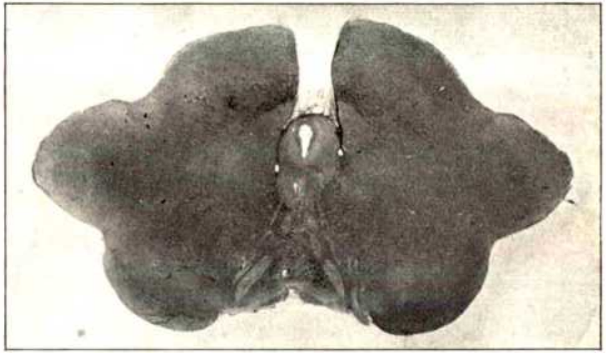

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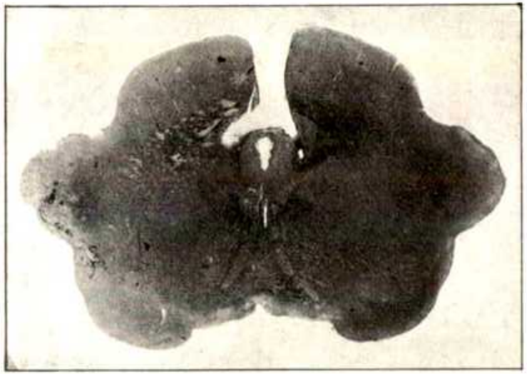

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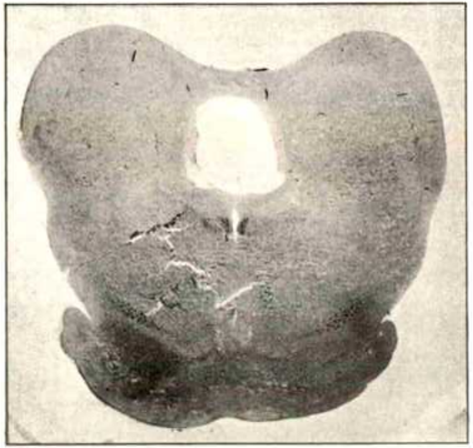

FIG. 4.

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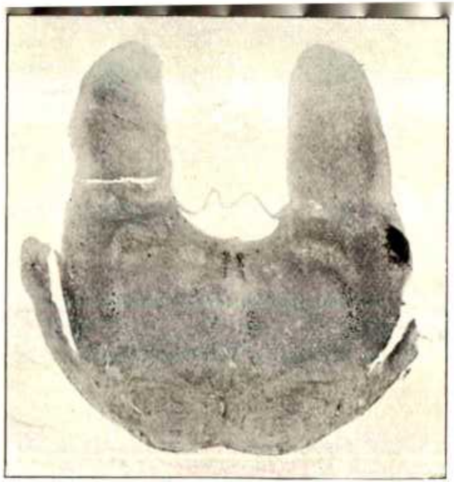

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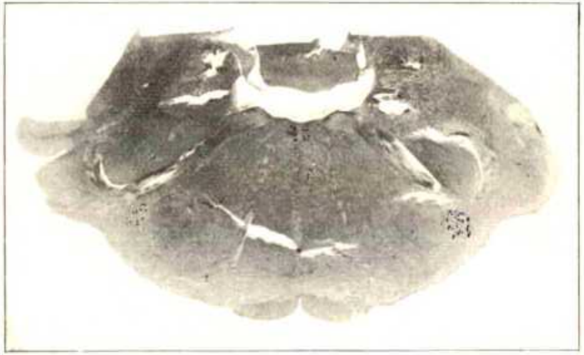

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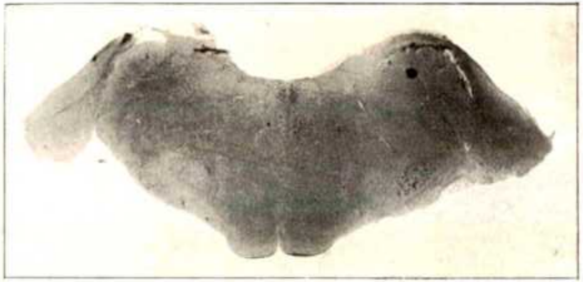

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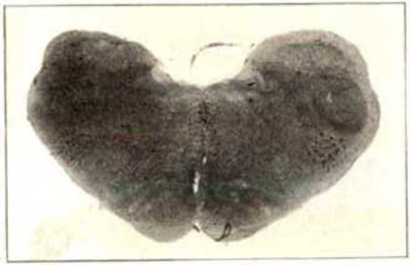

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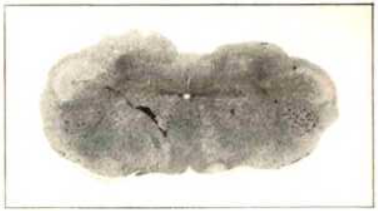
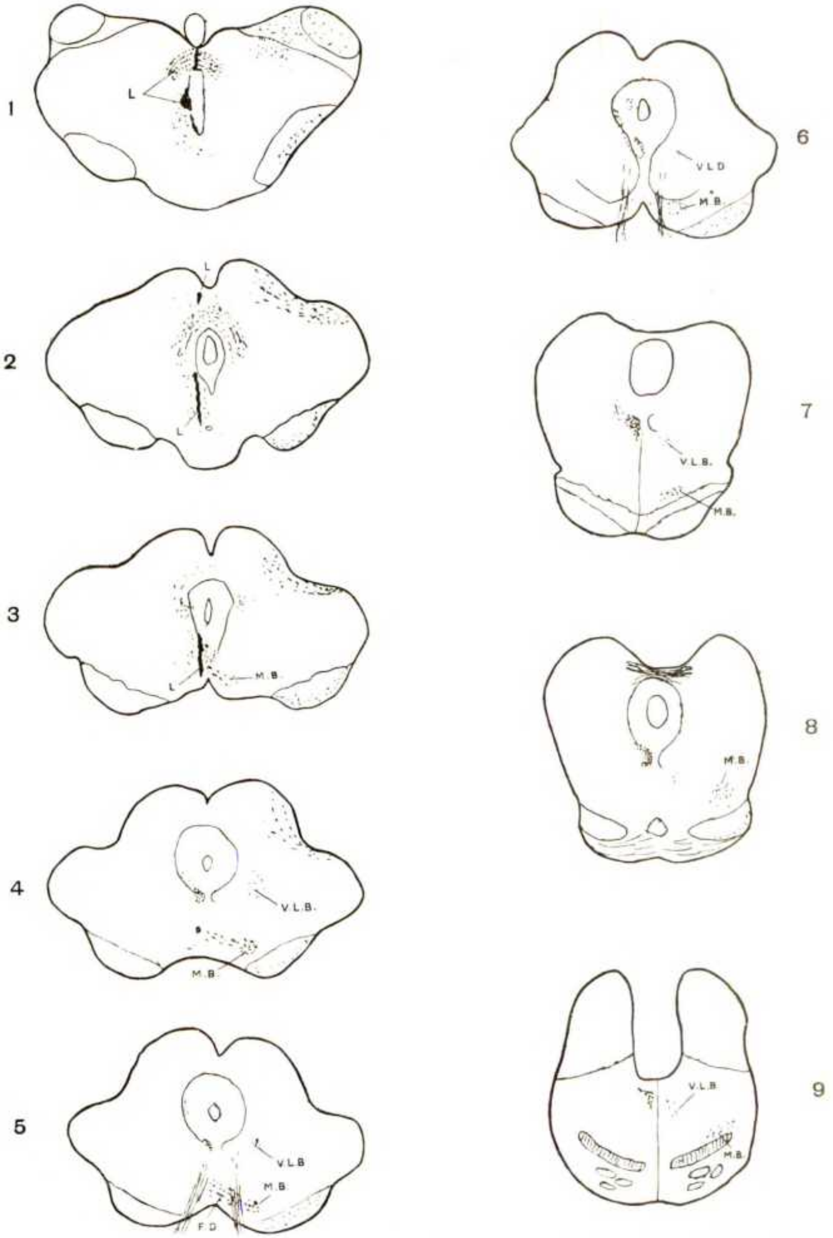


FIG. 9.

The same. Photographic reproductions.

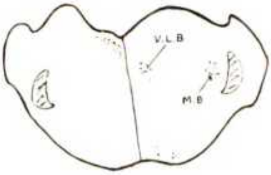
PLATE VI.



Cat. Sagittal linear lesion of mesencephalon to the right of the middle line. Degeneration below the lesion in right dorsal longitudinal bundle, left ventral longitudinal bundle and in the left Monakow's bundle. *Projection drawings.*

PLATE VII.

10



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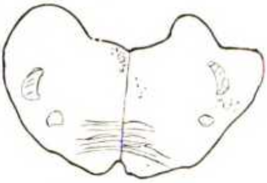

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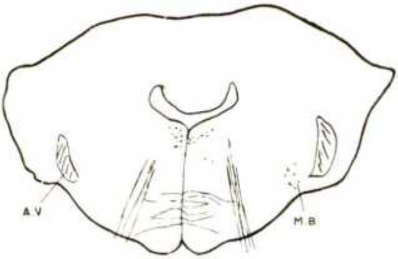

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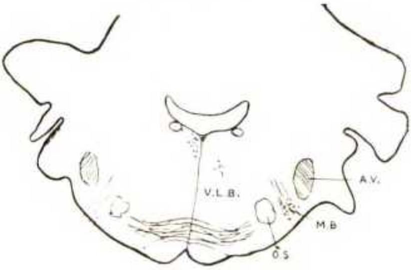

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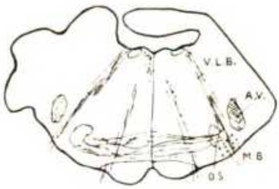

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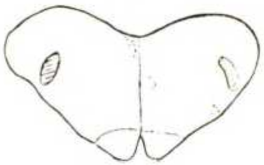

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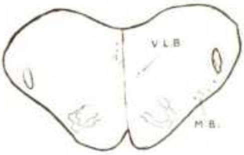

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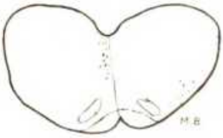

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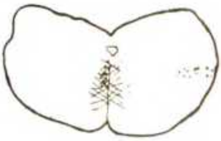

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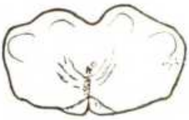

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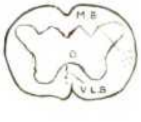

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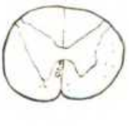

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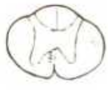

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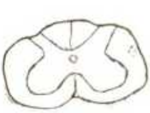
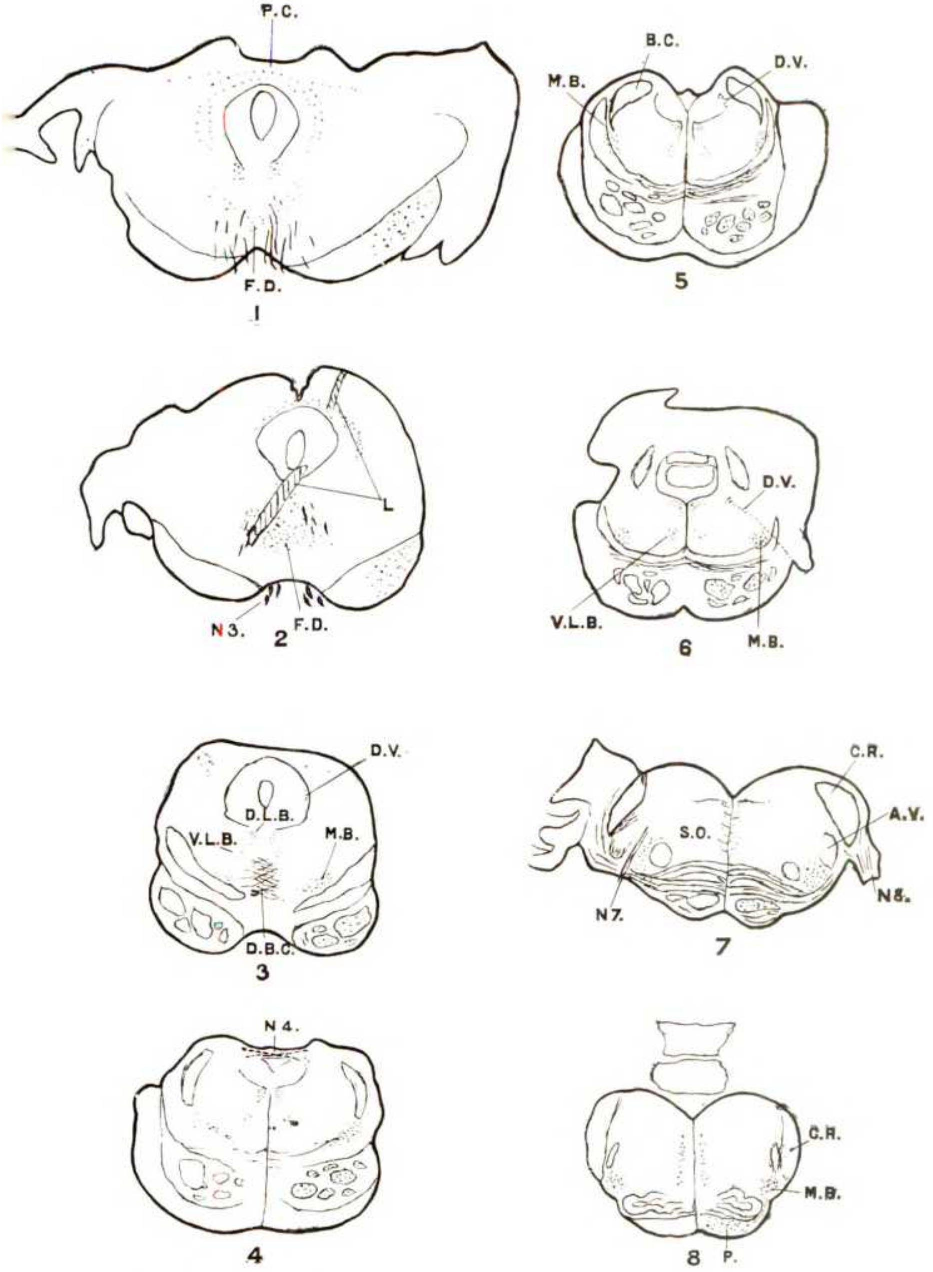
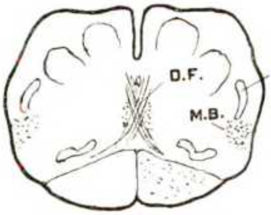


PLATE VIII.



Monkey. Lesion in R. superior colliculus running obliquely into region of left red nucleus. Degeneration in both Monakow's bundles, in both dorsal and ventral longitudinal bundles, in the right descending root of 5th nerve, and in one pyramid. *Projection drawings.*

PLATE IX.



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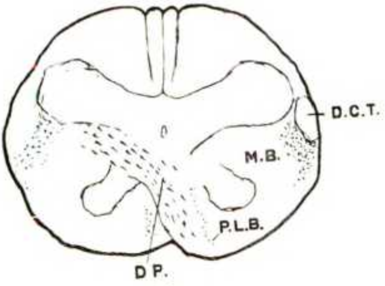

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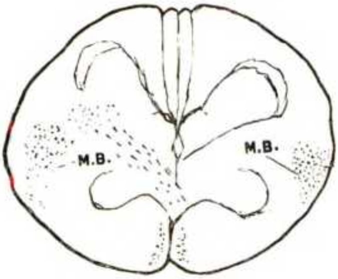

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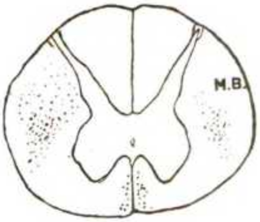

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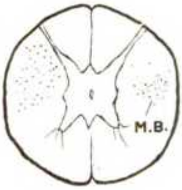

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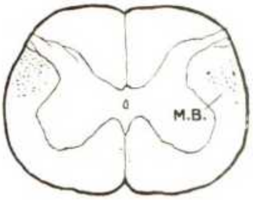

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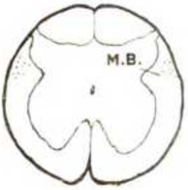

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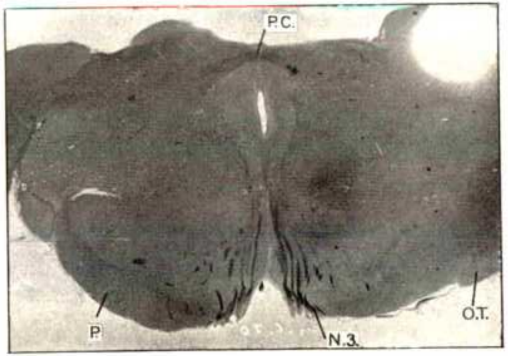
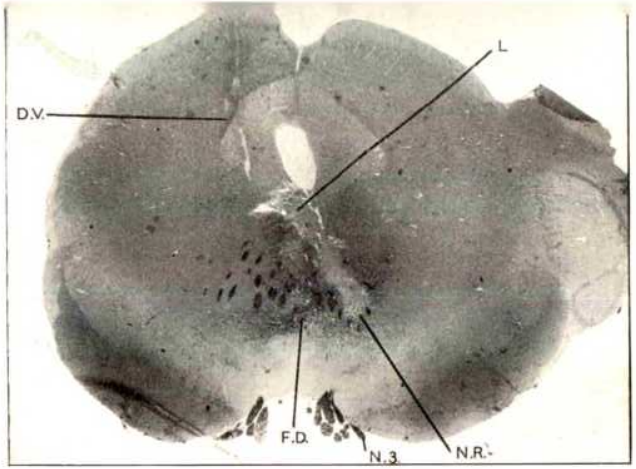


FIG. 1.



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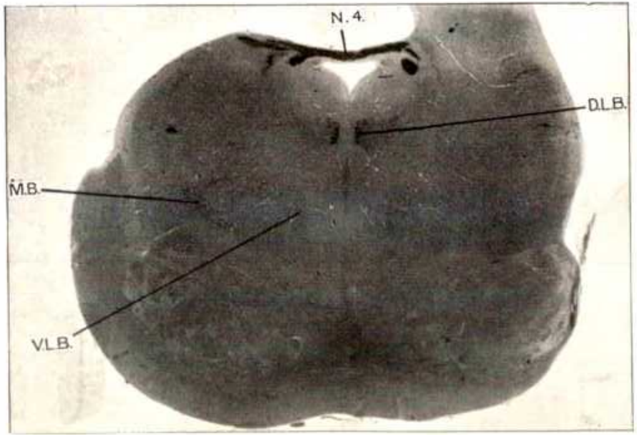

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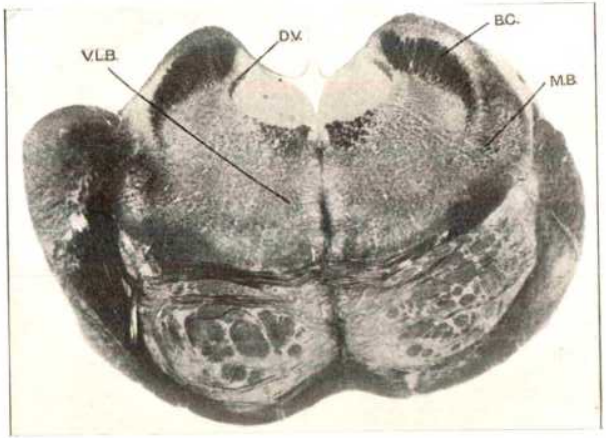
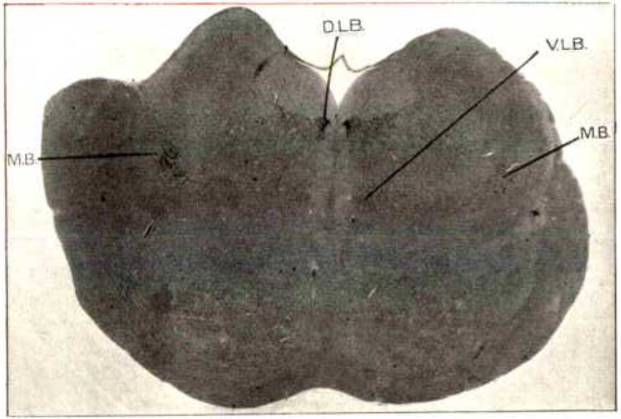


FIG. 4.

The same. Photographic reproductions.

PLATE XI.



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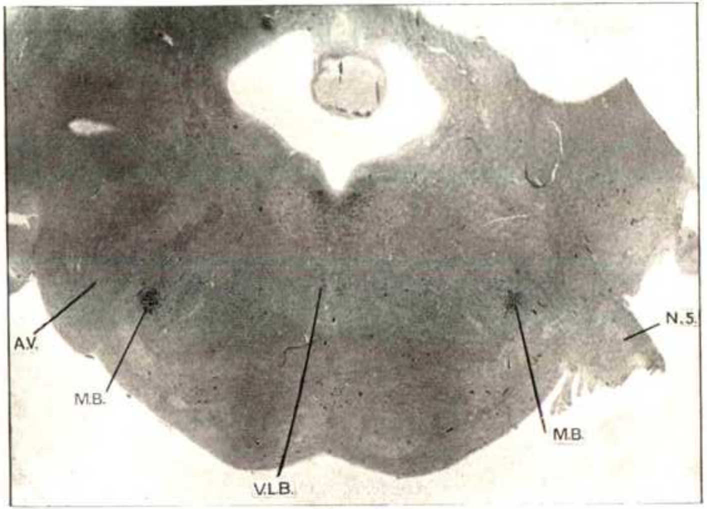

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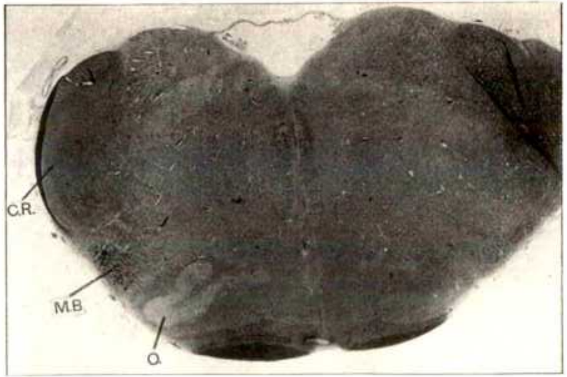

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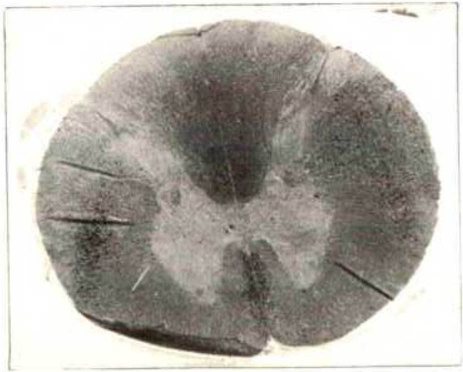


FIG. 8.

The same. Photographic reproductions.

PLATE XII.

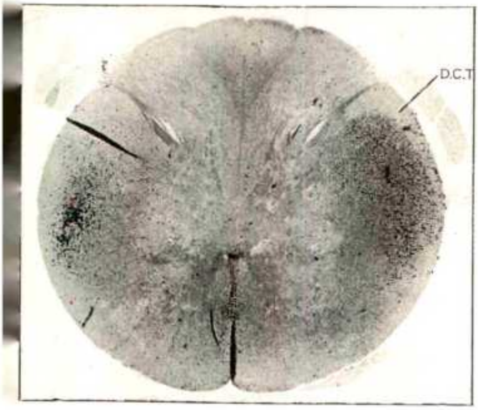
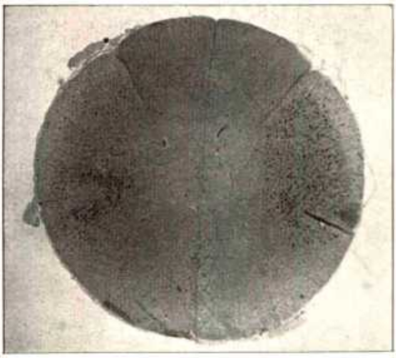


FIG. 9.



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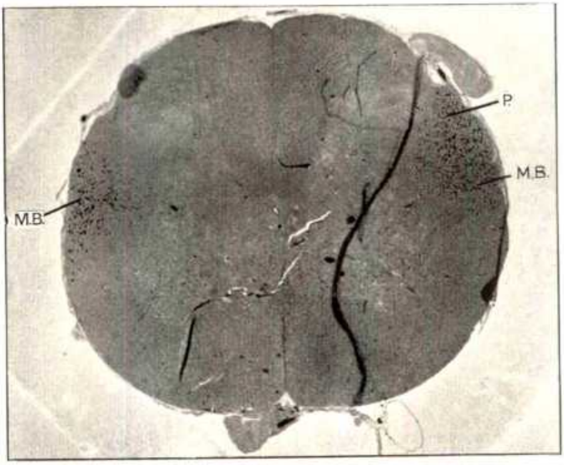


FIG. 11.

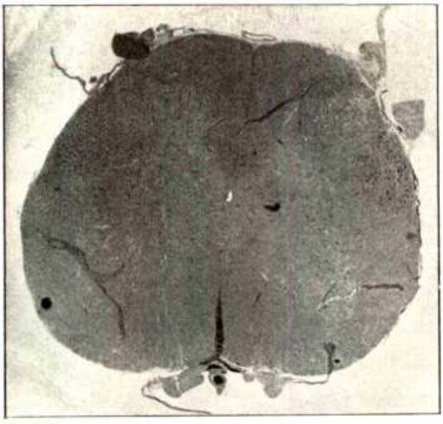


FIG. 12.

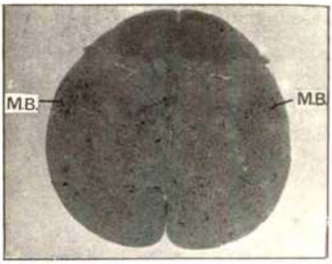
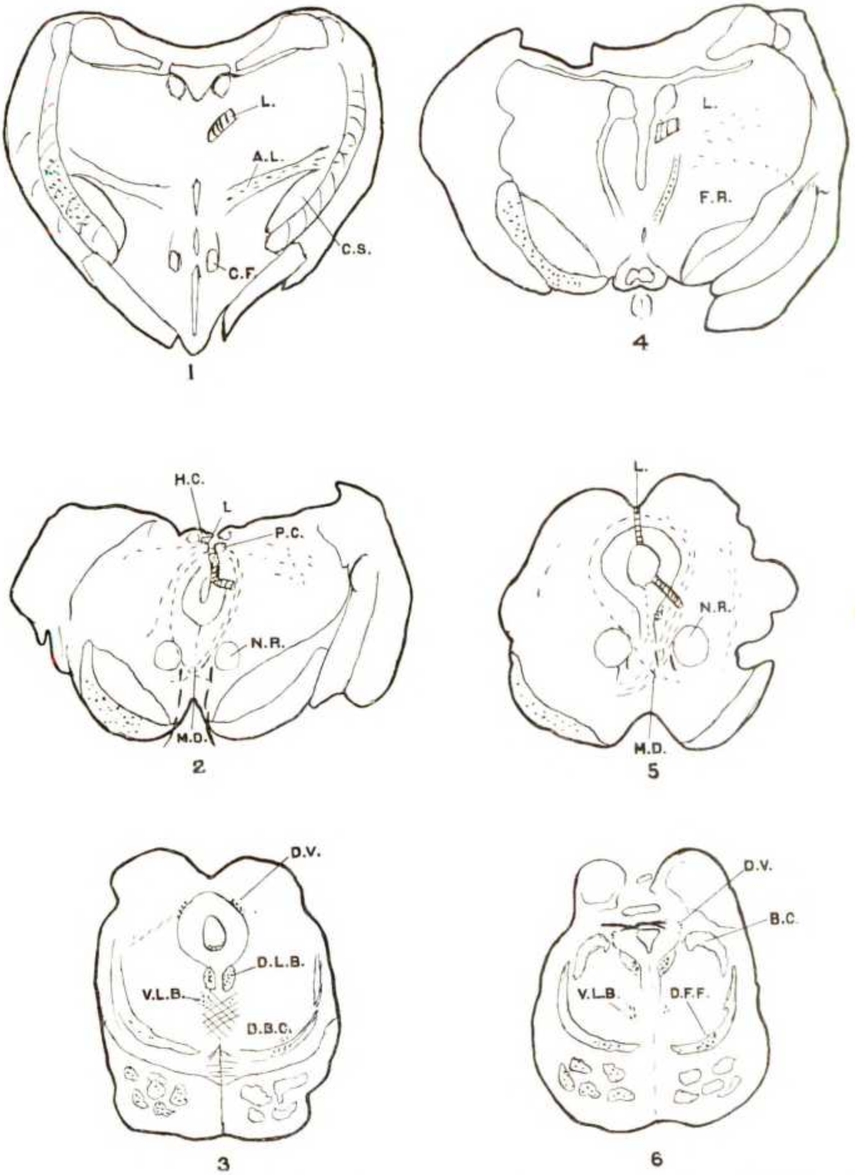


FIG. 13.

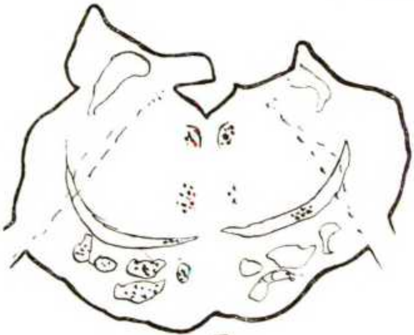
The same. Photographic reproductions.

PLATE XIII.



Monkey. Lesion extending from soft commissure backwards to the inferior colliculus, dividing the posterior commissure and destroying much of the gray matter around the Sylvian aqueduct. Degeneration in both dorsal and ventral longitudinal bundles, in both descending roots of 5th nerve, in descending fillet fibres on both sides and in one pyramid. *Projection drawings.*

PLATE XIV.



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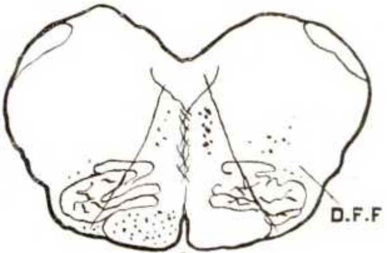

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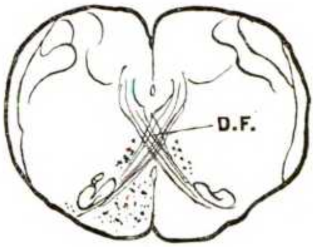

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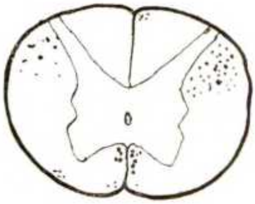

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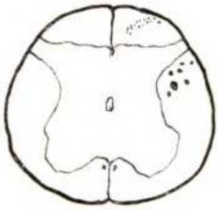

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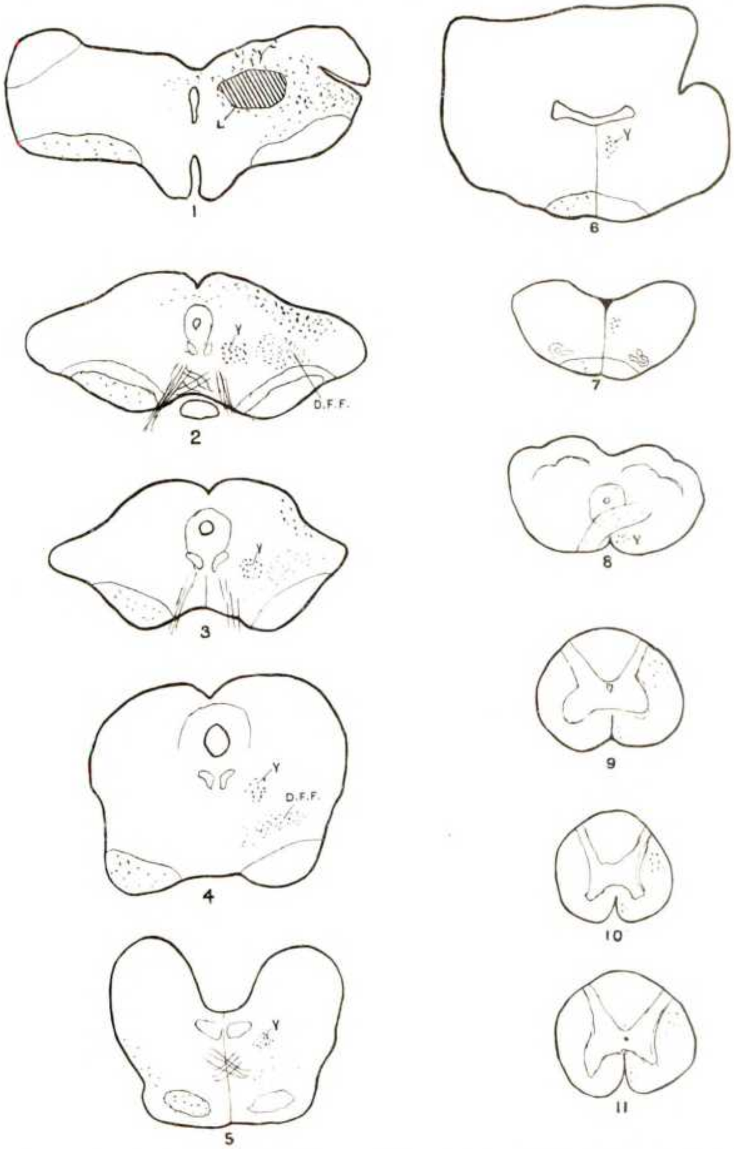
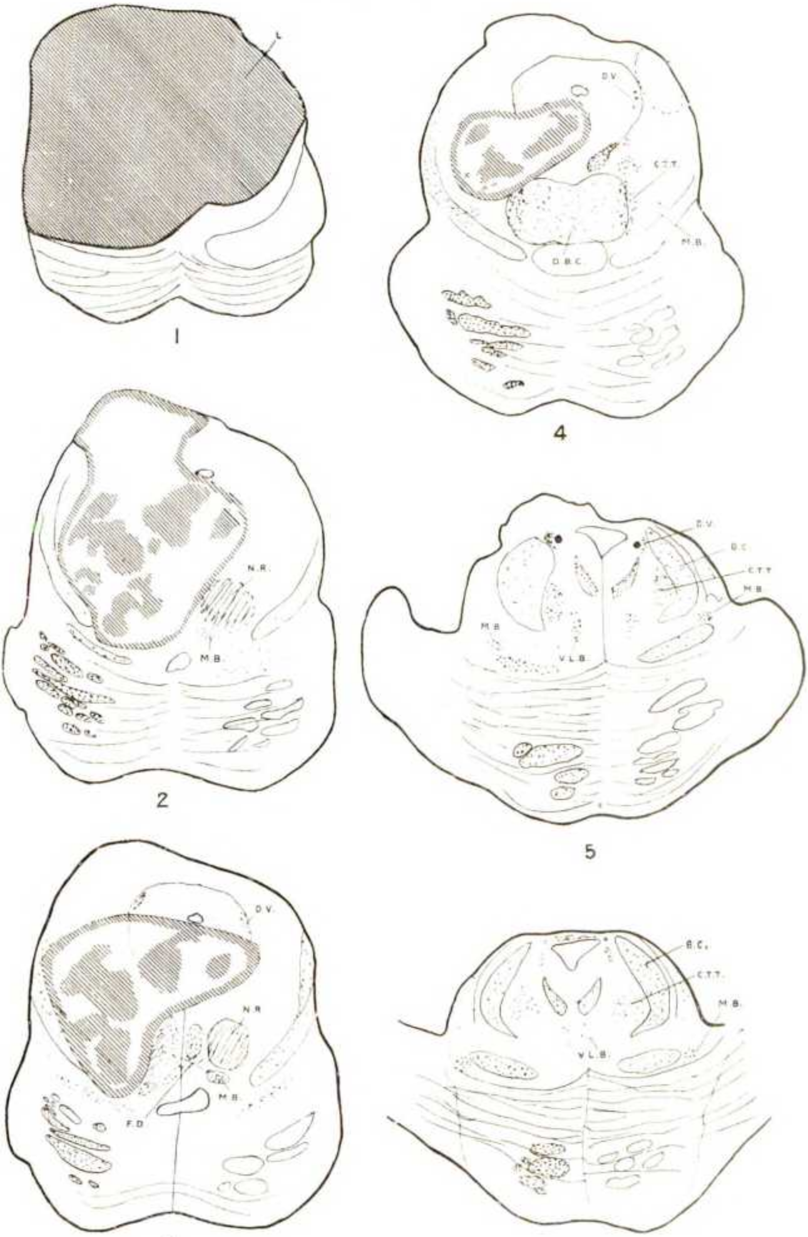
PLATE XV.



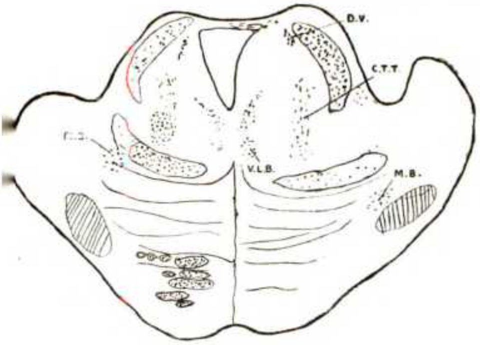
Cat. Lesion of right optic thalamus. Degeneration behind lesion in post commissure, and in a bundle lying immediately ventro-lateral to the dorsal longitudinal bundle of side of lesion and extending into the medulla and spinal cord, in stratum opticum and in upper fillet. *Projection drawings.*

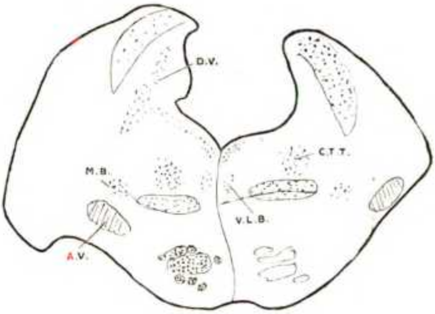
PLATE XVI.

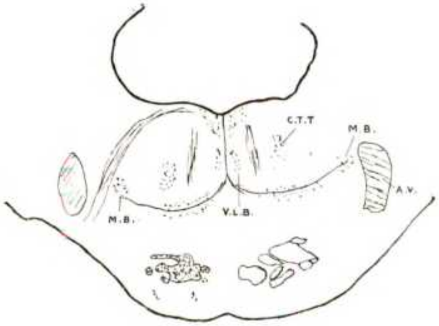


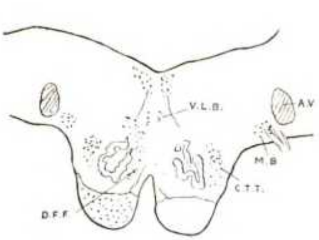
Tumour of mesencephalon. Descending degeneration in the left pyramid, both central tegmental tracts, both Monakow's bundles, both brachia conjunctiva, both descending trigeminal roots and in both mesial fillets. *Projection drawings.*

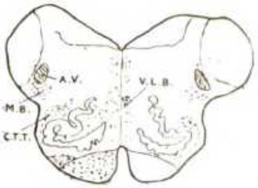
PLATE XVII.











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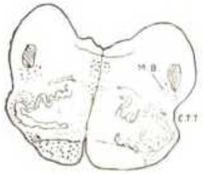

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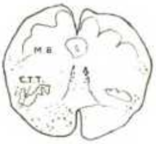

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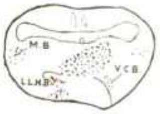

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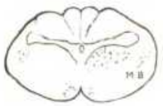

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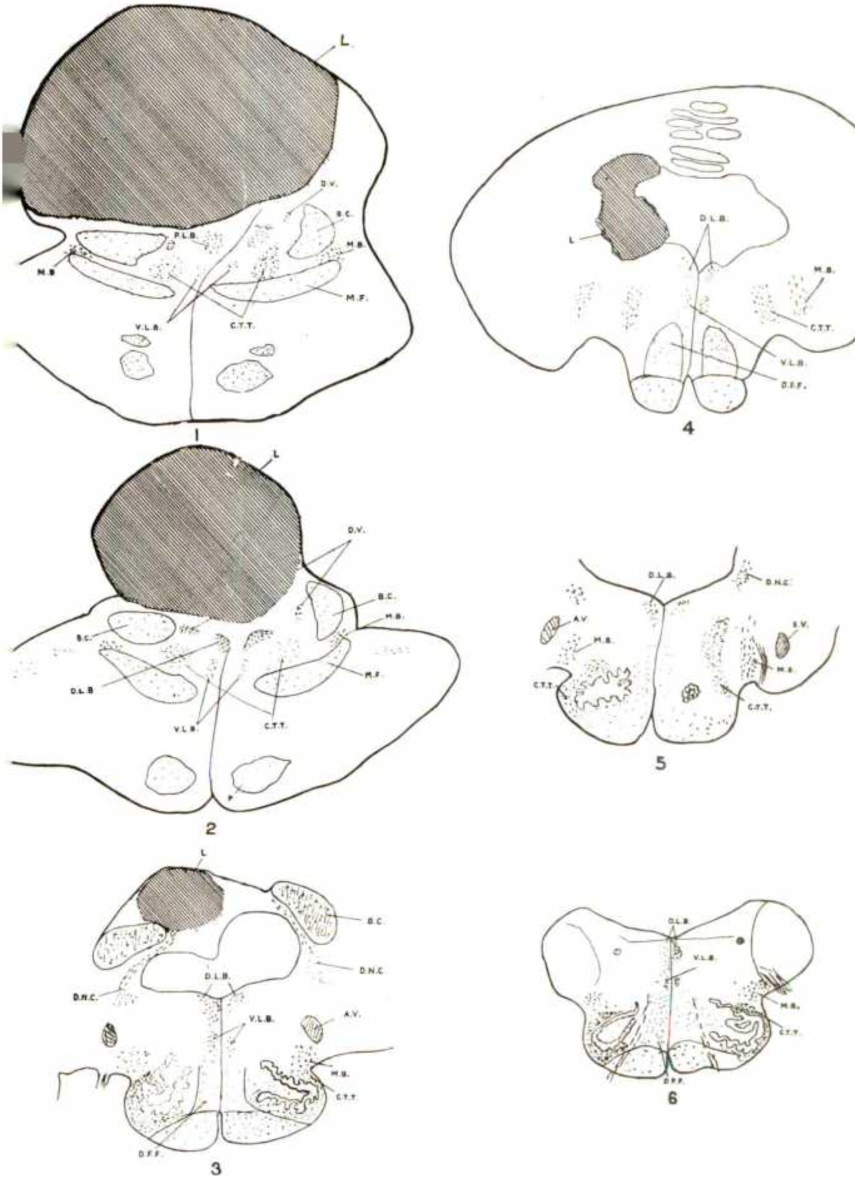
PLATE XVIII.

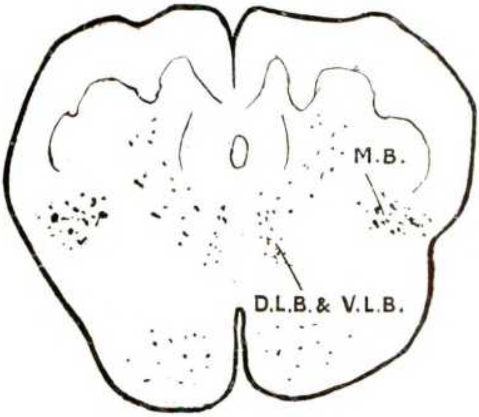


Abscess of the mesencephalon. Degeneration in both dorsal longitudinal bundles, both ventral longitudinal bundles, both central tegmental tracts, both brachia conjunctiva; both descending trigeminal roots, both Monakow's bundles and in both mesial fillets.

Degeneration in a tract on either side running from the superior medullary velum to Deiters's nucleus. *Projection drawings.*

PLATE XIX.

7



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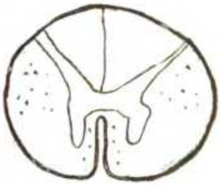

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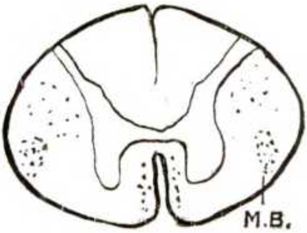

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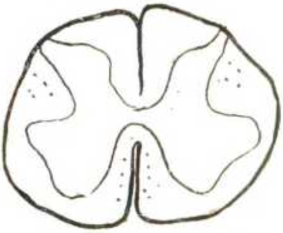

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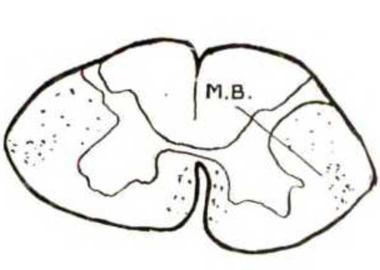
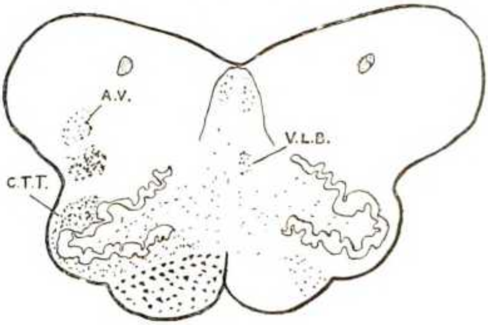


PLATE XX.

1



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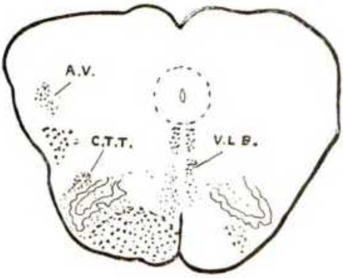

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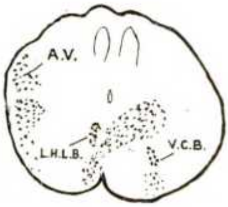

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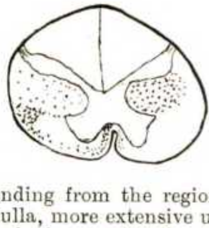
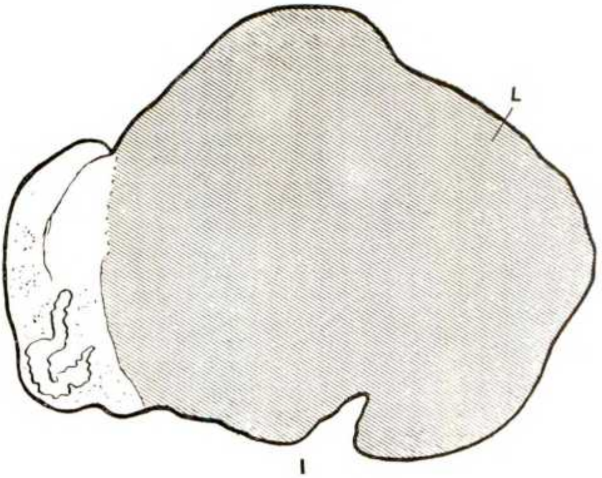


Tumour of the pons extending from the region of the inferior colliculi to the junction of the pons and medulla, more extensive upon the left side.

Descending degeneration in both pyramids L. > R., in the left mesial fillet and central tegmental tract, left fasciculus proprius of the medulla, in both dorsal and the R. ventral longitudinal bundle, L. spinal trigeminal root. Atrophy of the left inferior olive. *Projection drawings.*

PLATE XXI.



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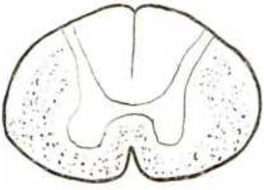

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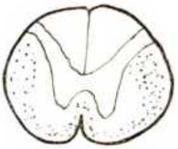

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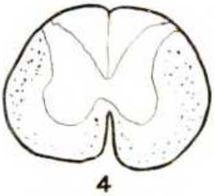
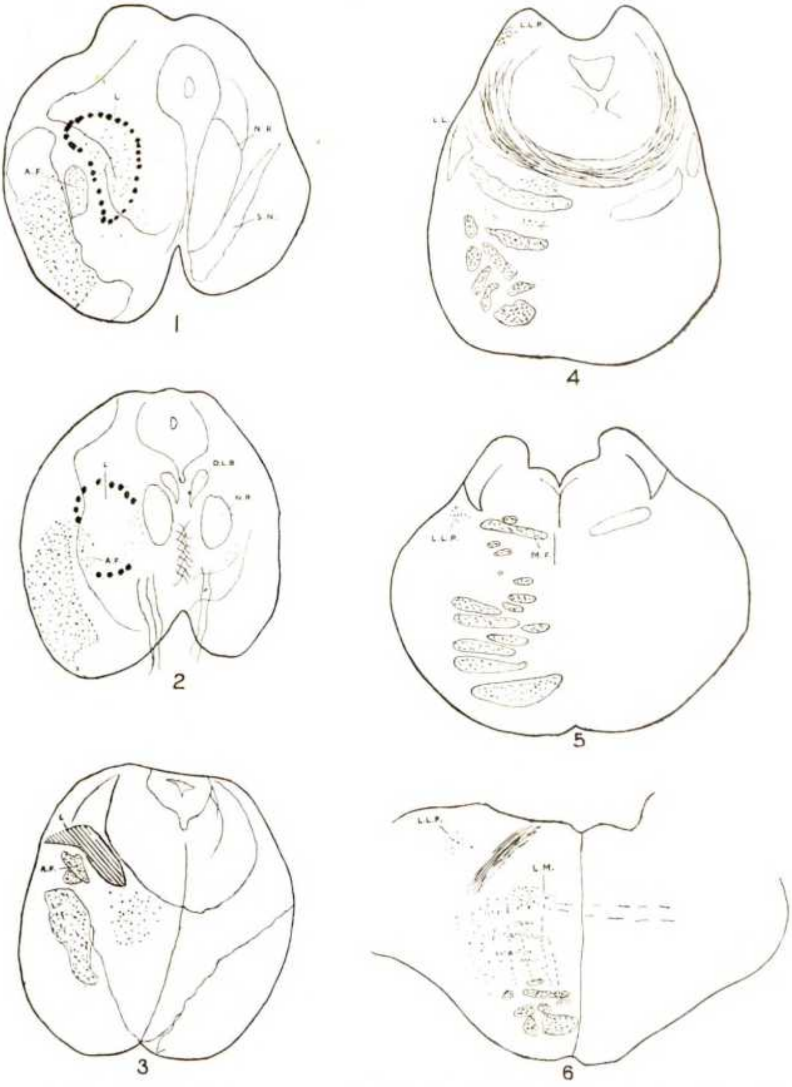


4

Tumour of the pons and upper medulla with practically complete destruction of the lower pons.

Degeneration in both ventral columns, both antero-lateral columns, and both lateral columns; the direct cerebellar tract and Gowers's tract alone escaping. *Projection drawings.*

PLATE XXII.



Tumour of left centrum ovale involving the left internal capsule. A localised patch of recent softening was present in the superior collicular region involving the nucleus lateralis superior, the upper part of the substantia nigra, the upper fillet and slightly the outer side of the red nucleus on the left side. A small hæmorrhage was present in the posterior inferior part of the left inferior colliculus.

Degeneration below the lesions in the left pyramidal tract, accessory fillet and mesial fillet.

From the lesion in the inferior colliculus a large fibred tract descended having a dorso-lateral situation in the pons and medulla, and in part joining the pyramid at the decussation, appeared to run as a ventro-lateral tract in the cord. *Projection drawings.*

PLATE XXIII.

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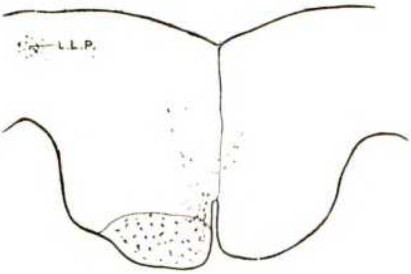

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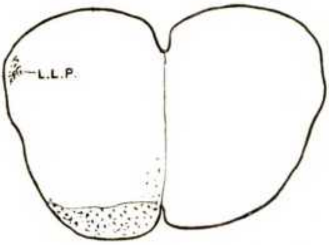

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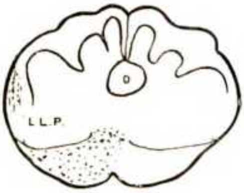

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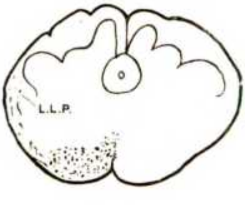

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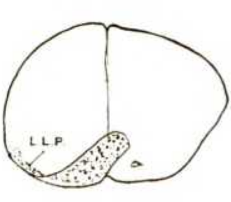

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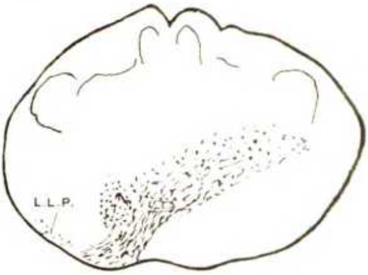

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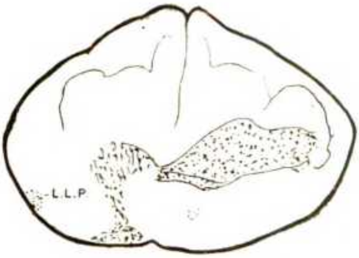

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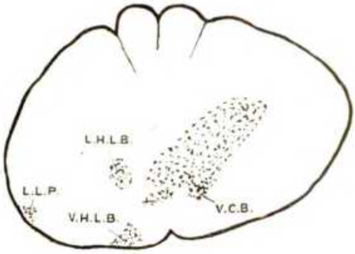

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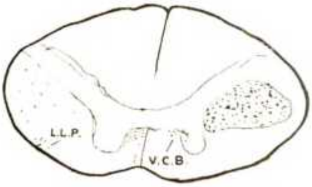
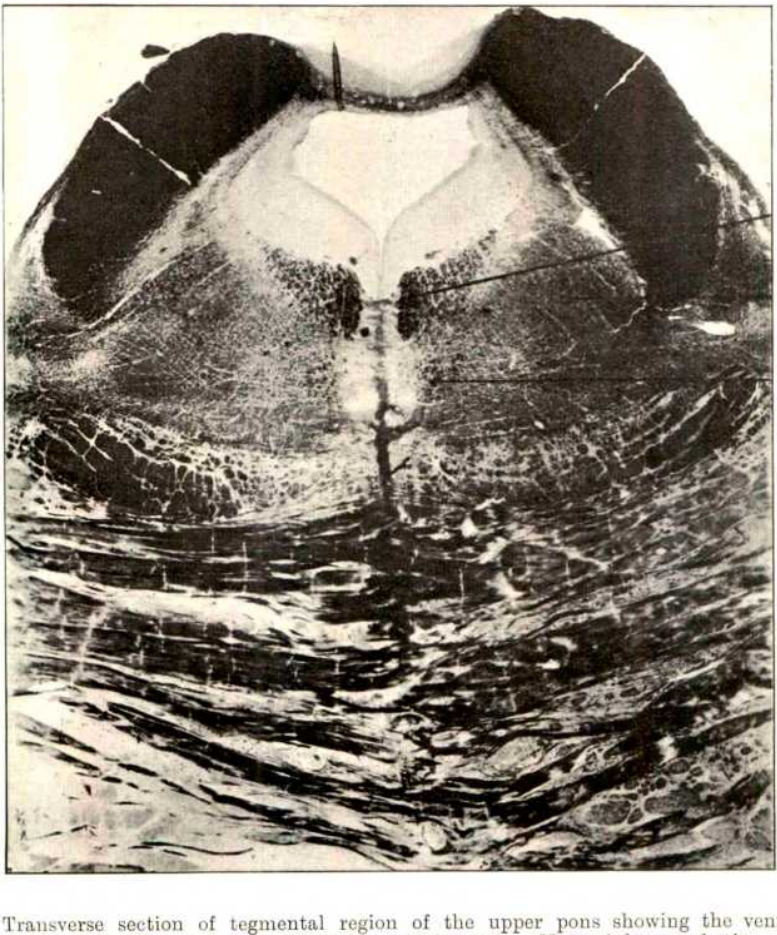


PLATE XXIV.



Transverse section of tegmental region of the upper pons showing the ventral longitudinal bundles and the central tegmental tracts. Normal human brain. Pal method.

described by Russell, *i.e.*, crossed and uncrossed ventral and lateral bundles. The course of the accessory fillet corresponded with Bechterew's and with Schlesinger's description except that many fibres appeared to rejoin the pyramid in the pons and medulla. Spitka's bundle (mesial accessory fillet) was not involved.

The tract of fibres from the region of the inferior colliculus is of peculiar interest and will be discussed later. Notwithstanding the lesion of the nucleus lateralis superior and red nucleus there was no degeneration in the ventral longitudinal bundle, nor in Monakow's bundle. Possibly this lesion was too recent to cause evident degeneration.

The foregoing observations bring forward little that is new, but they serve to bring together certain opinions of eminent investigators concerning the origin and destination of some descending mesencephalic tracts, which have hitherto been at variance. The presence of these tracts in man, which have been mostly investigated in animals, and their similarity of origin and course, have received but little attention in this country, and this fact will, we hope, justify the publication of these scanty observations preliminary to a more lengthy investigation upon this subject which it is our intention to pursue.

The points of interest in our observations may be summed up as follows:—

Monakow's bundle.—This tract, composed of large fibres, follows an almost identical course already described in the cat, monkey, and in man. It is relatively a large tract in the cat, relatively a small tract in man. So far as the descending fibres of this tract are concerned they appear to cross entirely in Forel's decussation. The majority of the fibres appear to have their origin in the caudal part of the red nucleus. Probst has recently found that wherever the red nucleus is injured degeneration in this tract follows, and that a lesion of a mesencephalon not involving the red nucleus gives rise to no degeneration of this tract. Our observations do not accord with his results. In experiment 5 and in case No. 5 the anterior and lateral aspect of the red nucleus respectively were injured and Monakow's bundle showed no degeneration. This suggests that the cells of the caudal part of the nucleus only give rise to the fibres in question. Further we have, beyond all doubt, traced fibres from the gray matter lying immediately ventro-lateral to the dorsal commissure, across Forel's decussation into Monakow's bundle and this in a case where there was no degeneration in the ventral longitudinal bundle of the crossed side. This origin for the fibres of Monakow's bundle was suggested by Held, Nissl and Boyce, while the majority of writers on the subject consider the red nucleus only to be the place of origin of the descending fibres of this tract. Our observations incline strongly in favour of a double origin for the fibres, the majority arising in the cells of the red nucleus, others in the gray matter close to the dorsal commissure. The latter fibres appear to cross just at the junction of Meynert's and Forel's decussations.

In more than one of our experiments a few fibres appeared to leave this tract as it lay in the fasciculus proprius medullæ and to run to the cerebellum with the restiform body. Such fibres have not, so far as we are aware, been described by other observers; they were the only fibres leaving the tract above the pyramidal decussation. In the cat, monkey, and man we have traced the tract into the sacral region, its termination in the ventral horns of the same side being easily seen. Only above the dorsal region were the fibres distinctly separated from those of the lateral pyramidal tract, lying in front of the latter.

The lateral ponto-spinal tract occupies apparently the same position in the medulla and spinal cord as does Monakow's bundle. Comparing the low pontine lesion in case 3 with the mesencephalic lesions in cases 1 and 2, the much greater degeneration in the fasciculus proprius medullæ and in the position occupied by Monakow's bundle in the cord in the former case is obvious. In this case the pontine nuclei and Deiters's nucleus of the left side were destroyed, and the large area of degeneration ventral to the spinal root of the fifth on the left side represents the combined lateral tract from the mesencephalon, pons, and upper medulla. Traced into the spinal cord, the degeneration in case 3 spread out in a band reaching from the lips of the ventral fissure nearly to the mid-point of the lateral column, and in the lateral column formed a wedge-shaped tract with base to the surface and apex stretching towards the lateral horn. Below the cervical region that part of the degeneration forming the wedge merged gradually into the lateral pyramidal tract (lateral ponto-spinal and rubro-spinal fibres), but the degeneration stretching along the surface outwards from the lips of the ventral fissure retained this position as long as it was traceable, that was to the lower dorsal region (vestibulo-spinal fibres).

The *ventral longitudinal bundle* is described as having its origin in the nuclei lateralis superior and centralis superior and as crossing entirely in the fountain crossway. We have not actually traced the degeneration from the nucleus lateralis superior, but have found marked degeneration of the bundle where this nucleus was intact. In most of our cases the fibres evidently arose from the gray matter immediately ventro-lateral to the dorsal commissure and having a curved course round the gray matter encircling the aqueduct, crossed in the fountain crossway. We can firmly support Held's statement that some of these fibres do not cross, but enter the ventral longitudinal bundle of the same side.

The crossed relation of the degeneration of the dorsal longitudinal bundle with that of the ventral longitudinal bundle and Monakow's bundle in unilateral lesions of the mesencephalon is striking, and is explained by the fibres of the dorsal longitudinal bundle being involved by such a lesion after they have crossed in the dorsal commissure.

The position of this tract as regards its proximity to the raphe of the pons and medulla seems to vary considerably. In the cord the fibres are spread out along the sides of the ventral fissure. We have traced them to the lumbar region.

The *vestibulo-spinal tract* must have been involved in cases 3 and 4, and the degeneration in the spinal cords of these cases involves an area amongst other degenerate areas quite corresponding with the classic description of the fibres descending from Deiters's nucleus to the spinal ventral horns. These fibres were traceable to the sacral regions of the spinal cord.

The above tracts, Monakow's bundle, the ponto-spinal tract, and vestibulo-spinal tracts, and some fibres of the dorsal and ventral longitudinal bundle, may be considered together as constituting a system descending from the various lower levels of the brain and probably constituting an alternative path by which motor impulses reach the spinal cord, the other path being the pyramidal system. In birds there is no direct descending path from the cerebrum, and, according to the investigations of Münzner and Wiener, Monakow's bundle is in these animals very well developed. In mammals the tracts here considered are well developed, it would seem, inversely to the development of the pyramidal system. In no case have we found so many fibres, for instance in Monakow's bundle in man, as in the cat. This inverse proportion of the development of these tracts and the pyramidal system is interesting in connection with the well-known persistence of voluntary movement after destruction of the pyramidal systems in lower mammals. Starlinger, Wertheimer and Le Page have shown that complete destruction of the pyramidal fibres at the decussation abolishes neither voluntary movement nor movements of the limbs as a result of cortical stimulation in the dog and monkey. Stoddart also proved that cortical excitation produced movements of the hind limbs and tail after longitudinal section of the decussation. It seems probable that the tracts here concerned represent the original paths for impulses from higher to lower parts in the nervous system, and that the path from cerebrum to cord, at first indirect, has with the process of evolution become direct by the development of a short circuit system—the pyramidal system—running direct from cerebral cortex to the lower levels. It has further been stated that no case has ever been brought forward in man in which complete destruction of the pyramidal tracts of one side below the thalamus has been followed by complete paralysis.

In connection with decerebrate rigidity and the presence of the deep reflexes in transverse lesions of the nervous system we may point out that when, as in Sherrington's original experiments, decerebrate rigidity is produced by mesencephalic transection in the cat, if hemisection of the medulla or cord be subsequently performed, such rigidity disappears at once on the side of and caudal of the hemisection. We suggest that the efferent paths essential for the production of decerebrate rigidity may be some of the tracts here concerned.

The integrity of the connection of the higher with the lower parts of the nervous system is now generally considered in man to be essential for the production of the deep reflexes. Total transverse lesions of the spinal cord abolish them. In connection with the disease "acute combined sclerosis" in which there is a progressive degeneration of all the long fibre systems in the spinal cord, the clinical aspect of the disease is, at first, one of spastic paraplegia, and subsequently if the patient live, one of flaccid paraplegia with absence of the deep reflexes. We would suggest, as the result of the pathological condition found by one of us (J. C.) in twelve cases, seven of which were published in this journal by Dr. Russell and Dr. Batten, that the onset of flaccid paralysis and loss of the deep reflexes is coincident with the degeneration of the remaining descending long fibres systems in the cord; for the pyramidal system suffers early in this disease and the short fibres systems have never been extensively affected. The afferent tracts are affected earliest of all in the disease and they have been found completely degenerate where spasticity continued to the end. It would seem to us then that the integrity of the long fibres systems of the antero-lateral regions of the cord dealt with in this paper, and which are among the last to be involved in combined degeneration is responsible for the maintenance of rigidity and the deep reflexes in default of the pyramidal systems and that with their degeneration flaccidity sets in and the deep reflexes are no longer obtainable.

We are promised the results of a wide experimental investigation into the physiology of these tracts at an early date by Rothmann.

It was pointed out as long ago as 1866 by Bouchard in cases of hemiplegia that the area of degeneration in transverse sections of the cord varied with the position of the lesion causing the hemiplegia. He showed that where the lesion implicated the hemisphere only the degeneration in the lateral column was sharply marked off ventrally, but that where the mesencephalon and pons were implicated the area of degeneration spread forwards towards the ventral roots and that the lower the lesion in the brain stem the more the degenerated area encroached upon the antero-lateral column. Many authors have since confirmed this fact, and indeed from the area of degeneration in sections of the cervical cord in such cases it is possible to infer with considerable accuracy the position of the lesion in the brain.

The *central tegmental tract* as degenerating caudalwards in lesions of the brain stem has been a most striking feature in three of the cases recorded here, and a few of its fibres were degenerated in one of the monkeys. In the other experiments the position and sagittal direction of the lesions probably explain the absence of degeneration of this tract.

We have found its course exactly as described by Bechterew, but our cases did not afford an investigation of its origin further than the fact that it was not degenerated in the cat in a lesion of the dorso-mesial part of the thalamus.

Most of the fibres of the tract appeared to end on the surface of the inferior olive of the same side, but some running between the pyramid and the olive appeared to enter the hilum olivæ and some seemed to be lost in the interolivary layer. As, however, in each of our cases where the central tegmental tract was degenerate, descending fibres of the fillet were degenerate in the interolivary layer, we were unable to trace with certainty the fibres of the central tegmental tract in the interolivary layer. We were unable to trace any of the fibres into Helweg's triangular tract.

In the *Neurologische Centralblatt* for June 16, 1901, Professor Obersteiner publishes a case of tumour. of the dorsal aspect of one half of the medulla in which he figures what he considers to be degeneration in the triangular field and traces this to the upper thoracic region of the spinal cord. He considers this tract to arise above the olive and to have no connection with the central tegmental tract which he considers to end in and upon the olive. We would point out, however, that the degeneration he figures corresponds exactly with that of the vestibulo-spinal tract, with perhaps some fibres of the rubro-spinal and ponto-spinal tracts. From his figure the former must have been implicated in the lesion, and the latter possibly. He figures a coarse fibre degeneration which, he admits, is not in the recognised position of the triangular path, and traces it to the dorsal region. The triangular path is, however, composed of very minute fibres. We cannot think that his interpretation of this case is clearly proved, nor that his dissent, on the grounds of his case, from von Bechterew's assertion upon the origin of the triangular path in the inferior olive (fasciculus periolivaris), logical.

In two of our cases there was marked atrophy of the inferior olive. This atrophy seemed to depend upon the degeneration of the central tegmental tract and not upon that of the fillet. This atrophy has been noted by Mott and others after hemi-transection of the brain stem and has been usually considered a disuse atrophy resulting from a lesion of the mesial fillet.

The descending fibres of the fillet seem to fall into at least three groups. Firstly, those fibres which descend doubtless from the pyramidal radiation from the accessory fillet of von Bechterew (lateral pontine bundle of Schlesinger) lie at first as scattered bundles in the substantia nigra, close to the dorso-mesial aspect of the crusta, and which subsequently enter the mesial fillet to be distributed to the nuclei of certain cranial nerves (coarse fibres of late myelination). Secondly, fine fibres of the mesial fillet which in the cat and monkey degenerate downwards after lesions of the thalamus and upper fillet, and can be traced for a certain distance only, apparently becoming lost in the reticular formations of the pons and medulla. Lastly, in old standing lesions at least, a great many fibres of the mesial fillet degenerate below lesions and can be traced through the interolivary layer, some to the olivæ and some through the fillet decussation to the posterior column nuclei. In cases where the mesial fillet has been destroyed the number of fibres which behave in this way is so great that the degeneration is plainly visible by the Pal method.

In case 5, the *accessory fillet* was entirely degenerated upon one side; it was traced from the capsule, and the degenerate fibres were seen to enter the motor nucleus of the fifth of both sides and the nucleus of the facial nerve on both sides and also the nucleus of the hypoglossal on both sides, but neither the nuclei oculo-motorii, abducentes, nor vagi. Many of these fibres seemed to re-enter the pyramid in the region of the medulla.

The fibres of the second class were met with in some of the experimental lesions and in case 5. Apparently they were fine fibres with origin above the mesencephalon, running down in the region of the upper and subsequently the mesial fillet, and becoming scattered and finally lost in the reticular formation of the pons and medulla.

The third group of fillet fibres were degenerated in all the cases where the middle fillet was injured. Such degeneration has been referred to by many authors as "retrograde degeneration." However, this degeneration may be most obvious in so short a time as eight weeks after the lesion, as we have noticed in more than one case in the human subject (not reported here), and is in no way that we are aware of to be distinguished from ordinary degeneration. It may be argued that this degeneration of the fillet is of the same nature as is the posterior root and posterior column degeneration which occurs in connection with cerebral tumour and vascular lesions of the brain, whatever the nature of the process. If such argument be valid the degeneration is an ascending one. The posterior degeneration is, however, independent of the location of the lesion, whereas the descending fillet degeneration is consequent only, so far as we are aware, upon an implication of the tract itself.

The tract descending from the region of the inferior colliculus in case 5 is peculiar. Its course, outside the superior brachium and restiform body in the lateral part of the pons and subsequently on the dorso-lateral aspect of the restiform body, was remarkable.

Moreover it was separated off anatomically in the region of the restiform body as a rounded bundle and could plainly be seen on the non-degenerate side. We have searched through a number of specimens of this region stained by Pal's method from different cases, and have as yet failed to find any similar tract separated off from the restiform body. It was composed of large fibres and in part seemed to join the pyramid just above the decussation, in part ran in the ventro-lateral region of the spinal cord. The fibres joining the pyramid seemed continued into the uncrossed ventral pyramidal tract.

We are unaware of any previous description of such a tract. From its mode of termination and from the fact that it was degenerated upon the side of a complete pyramidal degeneration, we would suggest that it was an aberrant pyramidal bundle, though its widely lateral course in the pons is not in line with this suggestion. From the position of the hæmorrhage in the inferior collicular region it was impossible to trace its upper connection with the pyramid if such existed.

The Tracts from the Nuclei Fastigii to Deiters's Nuclei.

The degeneration in case 2 shows that some of the constituent fibres are cerebellofugal. We were unable to trace any of these fibres to the nucleus of the vagus.

Fibres descending from the Thalamus to the Ventral Columns.

In experiment 5 (cat) a well-marked bundle of fibres descended from the thalamic region to the ventral column having an uncrossed course and being situated ventro-lateral to the ventral longitudinal bundle. They were not fibres of the fillet system, being always situated in a position ventral to the latter. They did not correspond with Held's uncrossed fibres of the ventral longitudinal bundle since their origin was in the deep part of the thalamus and their course not that of the ventral longitudinal bundle. Some of these fibres were traced in the ventral column of the cord close to the fissure to the lowest cervical region.

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EXPLANATIONS OF ABBREVIATIONS IN FIGURES AND PHOTOGRAPHS.

A. C. Q.—Anterior corpus quadrigeminum.
A. F.—Accessory fillet.
A. V.—Spinal root of 5th.
A. L.—Ansa lenticularis.
B. C.—Brachium conjunctivum.
C. T. T—Central tegmental tract.
C. S.—Corpus subthalamicum.
C. F.—Columns of fornix.
C. R.—Corpus restiforme.
D. L. B.—Dorsal longitudinal bundle.
D. B. C.—Decussation of brachia conjunctiva.
D. B. 6.—Fibres from dorsal longitudinal bundle to opposite 6th nucleus.
D. F. F.—Descending fillet fibres.
D. V.—Descending root of 5th.

D. F.—Decussation of fillet.
D. P.—Decussation of pyramids.
D. C. T.—Direct cerebellar tract.
F. D.—Forel's decussation.
F. R.—Fasciculus retroflexus.
H. C.—Habenular commissure.
I. C.—Internal capsule.
L.—Lesion.
L. F.—Lateral fillet.
L. H. B. L.—Lateral homolateral bundle of pyramid.
L. L. P.—Bundle from inferior collicular region to ventral column.
M. D.—Meynert's decussation.
M. B.—Monakow's bundle.
M. F.—Mesial fillet.
M. C.—Middle commissure.
N. 3.—Nervus oculo-motorius.
N. 4.—Nervus trochlearis.
N. 5.—Nervus trigeminus.
N. 6.—Nervus abducens.
N. 7.—Nervus facialis.
N. 8.—Nervus auditorius.
N. L.—Nucleus lenticularis.
N. L. M.—Nucleus lateralis medullæ.
N. R.—Nucleus ruber.
O.—Inferior olive.
O. T.—Optic tract.
P.—Pyramid.
P. C. Q.—Posterior corpus quadrigeminum.
P. C.—Posterior commissure.
S. O.—Superior olive.
Th. O.—Optic thalamus.
V. C. B.—Ventral crossed bundle of pyramid.
V. H. L. B.—Ventral homolateral bundle of pyramid.
V. L. D.—Ventral longitudinal bundle.
V. S.—Vestibulo-spinal tract.
Y.—Fibres from thalamus to ventral column.

DEGENERATIONS FOLLOWING A TRAUMATIC LESION OF THE SPINAL CORD, WITH AN ACCOUNT OF A TRACT IN THE CERVICAL REGION.

BY PURVES STEWART, M.A., M.D., M.R.C.P.

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The case upon which the following paper is based was that of a female patient, aged 28, in the Westminster Hospital, who sustained a dislocation of the vertebral column between the bodies of the 5th and 6th cervical vertebræ, crushing the spinal cord at the upper part of the 7th segment for a vertical extent of about half an inch. The patient survived for ten weeks after the injury. The clinical aspects of the case have been dealt with in a former article in this Journal.[1]

The whole spinal cord, the medulla oblongata and the pons Varolii were obtained for examination. Sections of the pons, medulla and cord were stained by the Marchi method, together with the anterior and posterior nerve-roots of both sides from the 5th cervical to the 1st thoracic inclusive. The segments of the cord in the vicinity of the lesion, both above and below, were stained by the Thionin-Nissl and by van Gieson's hæmatoxylin and picro-fuchsin methods; segments from the mid-thoracic and mid-lumbar regions were also examined by the latter methods.

I desire here to express my indebtedness to Dr. Ferrier and to Dr. Batten for their courtesy in permitting me to prepare the sections in the Neuro-pathological Laboratory, King's College, and in the laboratory of the National Hospital, Queen Square, respectively.

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[1] BRAIN, 1900, vol. 23, pp. 139-156.

EXTENT OF LESION.

At the upper part of the 7th cervical segment the cord was abruptly flattened to a thin ribbon, measuring only 1mm. in antero-posterior thickness. Below that level there were hæmorrhages within the cord, extending, on the right side, through the gray matter and in the ventral part of the posterior column as low down as the lower part of the 8th cervical segment, and on the left side to a similar situation as far as the lower part of the 7th cervical segment: no hæmorrhage extended upwards on the cephalic side of the lesion beyond the immediate vicinity of the injury. None of the nerve-roots, anterior or posterior, were torn across. On examination of the *anterior nerve-roots* on both sides, from the 3rd cervical to the 1st thoracic inclusive, the *7th cervical pair* were alone found degenerated. All the corresponding posterior nerve-roots of both sides were quite free from degeneration in their extra-medullary course, as shown by the Marchi stain.

EXPLANATION OF ILLUSTRATIONS.

In the case of the spinal cord it should be noted that the most caudal part of every segment, from the uppermost cervical to the coccygeal, was systematically chosen for examination. In the medulla and pons thin slices were examined at short distances from each other. In every instance the section was magnified ten times and traced by means of Edinger's projection apparatus and the degenerations accurately filled in, partly under the projection apparatus and partly by comparison with the aid of the microscope. In this way a complete series of figures was obtained, illustrating the degenerations which had supervened in the various tracts. The figures were again photographed down to their present size.

We shall consider the degenerations in the following order:—

DESCENDING DEGENERATIONS.

(*a*) Lateral columns.
(*b*) Anterior columns.
(*c*) Posterior columns

ASCENDING DEGENERATIONS.

(*d*) Posterior columns.
(*e*) Antero-lateral columns.

(*a*) *Descending Degenerations of Lateral Columns.*

(1) *Lateral pyramidal tract.*—This can be traced from the level of the lesion down to the lowest part of the coccygeal segment. At the level of the upper sacral region the number of its fibres rapidly diminishes, but there can be no doubt that it extends to the caudal extremity of the cord. Ludwig Muller[1] has recently maintained that no degeneration is ever found in the lateral columns below the 3rd sacral segment, and v. Bechterew[2] and Ziehen[3] state that the tract terminates about the 3rd or 4th sacral segment. Barker,[4] in his text-book, states that a certain number of fibres can be traced into the lowest portion of the sacral region, whilst Risien Russell[5] and Déjérine and Thomas[6] state that the tract extends to the most caudal extremity of the cord. Our sections are in entire agreement with the statements of the two latter observers.

As low down as the 6th thoracic segment we observe that the outermost fibres of the crossed pyramidal tract do not reach quite to the periphery of the cord. From the 7th thoracic segment, however, down to the coccygeal region, they are adjacent to the lateral surface of the cord. The innermost fibres of the tract throughout the greater extent of the cord lie close against the gray matter, but from the 5th lumbar segment downwards the tract gradually recedes outwards and lies at some distance from the gray matter (see plates 5 and 6).

(2) *Tract "X": ventro-external to the crossed pyramidal tract.*—In the 7th and 8th cervical segments, close to the periphery of the cord, a small sharply-defined, degenerated area can be clearly seen at a plane corresponding to the tip of the lateral horn, ventro-external to the crossed pyramidal tract and separated from the latter by a distinct space (see plates 5 and 7). It has a short course and cannot be traced below the 8th cervical segment. It is closely applied to the

surface of the cord at that portion corresponding to the tip of the lateral horn of the gray matter.

Various descending tracts, in addition to those of the pyramidal system, have been described in the antero-lateral columns of the cord. Thus the vestibulo-spinal or efferent antero-lateral tract, descending from Deiters's nucleus, figured by Ferrier and Turner,[7] Risien Russell,[8] Probst,[33] and others, occupies the peripheral portion of the ventral part of the cord (see figure 1). But our tract is much smaller and more compact than the above, and lies farther back, in the lateral column, not in the anterior column as in the case of the vestibulo-spinal tract. Moreover, "tract X" stops short at the lower end of the cervical enlargement, whereas the vestibulo-spinal tract extends caudally as far as the lumbar region.

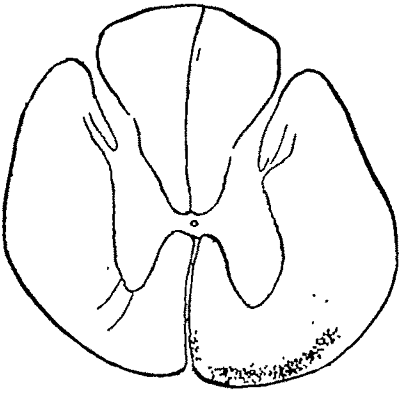


FIG. 1.

Descending tract from Deiters's nucleus. (Probst, *op. cit.*, Tafel vii., fig. 7.

The position of "tract X," sharply marked off by a distinct gap from the crossed pyramidal tract, also differs from that of the rubro-spinal tract described by Monakow,[39] in the dog, and by Boyce,[9] Held[30] and others in man, which is diffusely scattered through the antero-lateral column and overlaps the area of the crossed pyramidal tract (see figure 2).

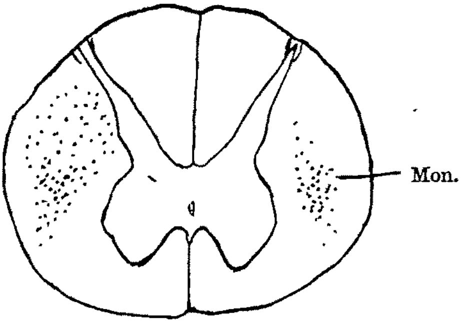


FIG. 2.

Monakow's bundle in the monkey, after a mesial lesion in Forel's decussation. (From a drawing by Dr. James Collier.)

The "triangular pre-pyramidal tract," described by André Thomas[34] in the cat in a case of hemisection of the cord (see figure 3) which is apposed by its base to the crossed pyramidal tract, whilst its apex is directed forwards, is probably identical, as Thomas admits, with Monakow's bundle, and, like it, can be traced to the lumbar region of the cord.

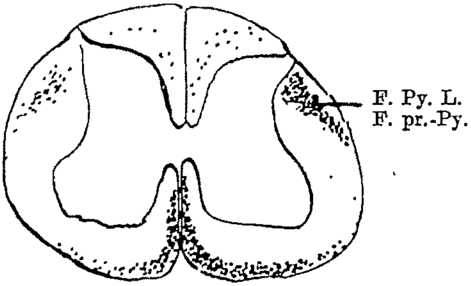


FIG. 3.

"Faisceau triangulaire pre-pyramidal," described by André Thomas (*op. cit.*, p. 49, fig. 3). Level of 7th cervical segment (cat).

(Monakow's bundle is more fully discussed by Buzzard and Collier in another article in this number of BRAIN, to which they have courteously allowed me access while their paper is going through the press.)

The descending tract from Bechterew's nucleus reticularis in the pons and which, in the cord, is situated close to the anterior median fissure—"Fissurenstrang" of Tschermak—only requires to be mentioned to show that it is in no way related to the tract now under consideration.

A tract corresponding somewhat more closely in position to the "tract X" is that described by Helweg[10] as the "triangular tract," and independently by von Bechterew[11] as the "olivary tract." Helweg's tract, however, lies more anteriorly than "tract X" (see figure 4). It has been a disputed point whether Helweg's tract is centrifugal or centripetal; Obersteiner[25] admits that the evidence is inconclusive one way or the other; Reinhold[12] claims to have traced Helweg's tract, although only by the Weigert-Pal method, in a case of melancholia, to the mid-thoracic region; but Bruce,[31] in his recent atlas, does not find it below the 3rd cervical segment, and Meyer[13] also traces it only to the 3rd cervical segment, after hardening by Müller's fluid, in a case of pontine hæmorrhage. Both Reinhold and Meyer incline to the view that it is a descending tract, which is suggested by Reinhold to be vasomotor in function. On the other hand, von Bechterew,[11] after originally stating that Helweg's tract arises "undoubtedly" from the cells of the lateral horn, proceeds in a more recent paper[11A] to explain that this is

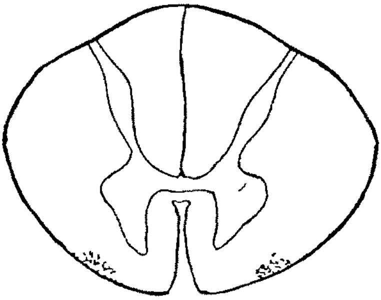


FIG. 4.

Helweg's tract at the level of the 3rd cervical segment. (From "Bruce's Atlas of the Spinal Cord.")

merely a "topographical" expression, and that the tract is really a descending one. Edinger[14] regards Helweg's tract as an ascending one. "Tract X," however, is a descending one, and it stops short at the lower end of the cervical enlargement. Further, its position, at a plane corresponding to the tip of the lateral horn, differs from that of Helweg's tract, which lies among the anterior nerve roots. Moreover, its degenerated fibres do not differ in average calibre from those of the crossed pyramidal tract, whereas both Helweg and von Bechterew agree that the "Olivenstrang" or "Dreikantenbahn" is made up of very fine fibres. Probably, however, the recent degeneration in our case renders the measurement of its individual fibres of less importance, since nerve-fibres may become considerably swollen during the process of degeneration.

On the whole, then, we may conclude that "tract X" is a descending tract which does not correspond in position to the "Dreikantenbahn" or "Olivenbündel" of Helweg and von Bechterew, and that, unlike the latter, it terminates at the lower end of the cervical enlargement.

The possibility of "tract X" being an aberrant strand of the pyramidal system must also be borne in mind. In a case such as ours, however, such a hypothesis is incapable of proof or disproof.

(*b*) *Descending Degenerations of Anterior Columns.*

(1) *Ventral pyramidal tract.*—This can be traced as a distinct band of degeneration, bounding the anterior median fissure in its whole depth, as far caudally as the 3rd lumbar segment. Below that level the ventral fibres of the tract become scanty and the part which extends farthest in a caudal direction is that portion which lies close to the bottom of the fissure. This latter portion of the tract can be traced as low as the lower part of the 4th sacral segment, below which level, although there are a few scattered degenerated fibres in the anterior columns, they can scarcely be dignified any longer by the name of a distinct "tract" (see plate 6).

This tract is said by Barker[4] and by Ziehen[15] to end usually in the mid-thoracic region, though both of these authors admit with Tooth[16] that in rare cases it may be followed to the middle of the lumbar enlargement. Dejerine and Thomas,[6] however, trace it to the 4th sacral and Risien Russell[5] to the 5th sacral segment. Our case is again in agreement with the three latter observers.

(2) *Efferent ventro-lateral tract.*—The fibres of this tract are known to be derived from various sources. Upon the discussion of these sources, the present case, being due to a bilateral spinal lesion, throws no light. The tract is seen to occupy the extreme periphery of the ventral portion of the cord from the anterior median fissure outwards. As low down as the 4th lumbar segment it is seen to be in close apposition with the ventral pyramidal tract. Below that level, as the ventral pyramidal tract shrinks towards the deepest part of the anterior median fissure, the efferent ventro-lateral tract remains close to the surface of the cord and can be traced as a distinct path as far as the lower part of the 4th sacral segment. Below the 4th sacral, scattered degenerated fibres are diffusely dotted over the whole ventral part of the cord.

As we trace this tract downwards it extends less and less outwards towards the lateral aspect of the cord; the part which extends farthest caudally being that which lies near to the anterior median fissure (see plate 6).

(3) *Fibres in anterior commissure.*—Degenerated fibres can be traced in the "commissura alba" for three segments below the lesion, as far down as the lower part of the 1st thoracic segment. Below that level the anterior commissure is free from degeneration.

(*c*) *Descending Degenerations of Posterior Columns.*

"*Comma*" *tract* of Schultze; *septo-marginal tract* of Bruce and Muir; *centrum ovale* of Flechsig and *triangle médian* of Gombault and Philippe (see plates 5 and 6).

The "comma" tract of Schultze forms a well-marked stripe of degeneration in the postero-external column of each side, extending from the dorsal aspect of the gray commissure in front to the posterior surface of the cord behind. This appearance of the tract is maintained, though its fibres gradually diminish in number from above downwards, as far caudally as the 9th thoracic segment. Below this level, the ventral part of the tract disappears, whilst its superficial, most dorsal portion continues to run on caudally as the "septo-marginal" tract, whose fibres at the level of the 10th thoracic segment begins to bend inwards, along the dorsal surface of the cord, towards the median septum. At the 11th thoracic segment they form a thin layer bounding the dorsal surface of the postero-internal column. In this position they run caudally for three segments (Th. 11 and 12 and L. 1). At the 2nd lumbar segment they turn the corner and dip ventrally inwards into the posterior median septum. At the 3rd, 4th and 5th lumbar segments the fibres lie close to the median septum, and at a little distance from the dorsal surface, the tracts of the two sides here forming the "oval field" of Flechsig. At the 1st sacral segment the dorsal margin of the tract again reaches the surface, and from this level it can be traced in a caudal direction down to the lowest part of the coccygeal region. From the 3rd sacral segment downwards the tracts of the two sides form a triangular area (*triangle médian* of Gombault and Philippe) whose base is situated at the dorsal surface and whose apex extends for a short distance into the posterior median septum.

Hoche, in his classic paper,[17] states that the "comma" tract is entirely separate and distinct from the combined bundle composed of the septo-marginal tract, the centrum ovale, and the triangle median; in which view he is supported by Barker,[18] who designates them as "two distinct and now fairly sharply defined descending tracts," and by Dejerine and Theohari,[19] who also regard Schultze's comma tract as an entity anatomically independent of Hoche's peripherally situated bundle. Our case, however, tends to show that no line of demarcation can be drawn between these various bundles of descending fibres, and that they may reasonably be considered as forming, not two, but one long continuous system. Nageotti and Ettlinger,[28] from a study of two cases of transverse lesion in the dorso-lumbar region, also come to this conclusion, which is also supported by Janischewsky's[32] case of lesion in the 7th cervical segment. The question as to which fibres of this system are endogenous and which exogenous in origin is not elucidated by our case, inasmuch as the lesion was one which destroyed both endogenous and exogenous fibres at the level of injury.

(2) *Short descending fibres near median septum.*—In addition to the combined system of tracts already described, our sections show, as in Hoche's case, another group of descending fibres in the dorsal funiculi of the cord, lying along the posterior median septum about its middle, but not extending farther down than the 8th cervical segment. These fibres are evidently short ones, distinct from those of the "comma" tract.

Finally, a number of degenerated fibres lie scattered diffusely over the whole field of the posterior columns, and most abundantly near the dorsal surface of the postero-internal columns. They also stop short below the 8th cervical segment, and may probably be fairly included within the limits of "traumatic" degeneration.

(*d*) *Ascending Degeneration of Posterior Columns.*

At the 5th cervical segment, the posterior columns show well-marked degeneration over their entire area, except in a limited portion of the postero-external column in the vicinity of the posterior horn where a number of undegenerated fibres are situated, having entered the cord through the posterior nerve-roots of the 6th cervical segment, above the level of the lesion. With this exception, the degeneration occupies the whole area of the posterior columns from the dorsal surface to the gray commissure, forming a wedge-shaped area whose apex points ventrally and is embedded in the concavity of a crescentic area of degeneration situated close to the gray matter—the "cornu-commissural zone" of Marie. Above the 2nd cervical segment this cornu-commissural crescent disappears, and at the lower end of the 1st cervical segment the wedge alone survives, its apex touching the gray commissure. The base of the wedge at this level lies both in the postero-external and postero-internal columns: its apex lies in the postero-internal columns only (see plate 4).

Sections through the lower and upper parts of the pyramidal decussation and through the lower end of the inferior olive show the blunting of the apex of the wedge by the gradual pushing backwards of the gray matter towards the dorsal surface, and the appearance, first of the nuclei graciles and then, at a higher level, of the nuclei cuneati, into which nuclei, but especially the cuneate, most of the degenerated fibres are traced (see plate 3).

But the degeneration does not cease abruptly at the posterior column nuclei. Distinct degeneration can be seen higher up, in the arcuate fibres of the medulla, both in the postero-external arcuate fibres, on the surface of the medulla, and in the decussating internal arcuate fibres. By the time the level of the lower end of the 4th ventricle is reached this degeneration of the arcuate fibres is less conspicuous and is seen chiefly in the vicinity of the raphé. In this respect our sections entirely corroborate the results of Hoche,[17] Schaffer[20] and others, although both Mott[23] and Sherrington[24] in experimental degenerations produced in monkeys failed to find degenerated fibres above the posterior column nuclei.

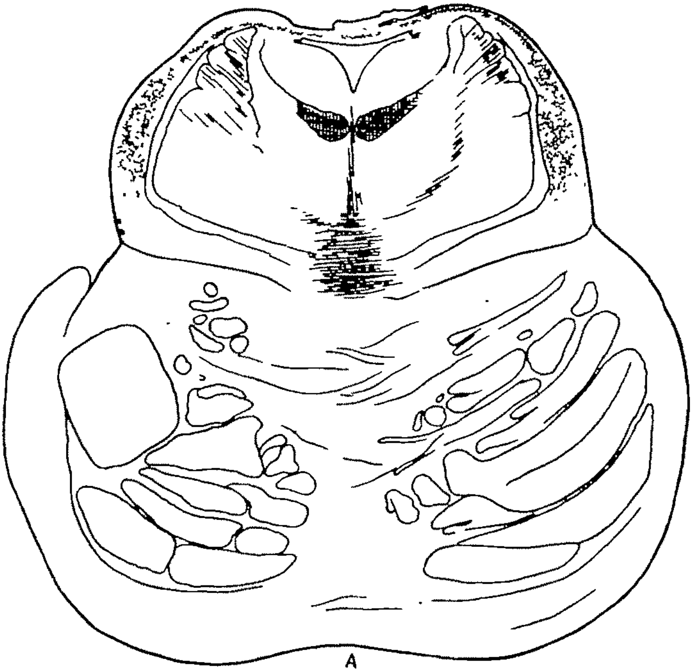
(e) *Ascending Degeneration of Antero-lateral Columns.*

(1) *Dorso-lateral, or direct, cerebellar tract.*—At the 5th cervical segment this forms a flat narrow strip at the periphery of the dorsal part of the lateral column. Its anterior margin abuts closely on the adjacent ascending antero-lateral tract, which is also degenerated. At the level of the pyramidal decussation the dorso-lateral cerebellar tract comes to form a somewhat wedge-shaped area occupying the angle between the substantia gelatinosa and the lateral surface of the cord. As we trace it higher, its fibres recede still farther dorsally and run external to the substantia gelatinosa to enter the corpus restiforme and go to the cerebellum (see plates 3 and 2).

(2) *Ascending antero-lateral tract* (*Gowers*).—The head of the "comma," situated dorsally, close to the previous

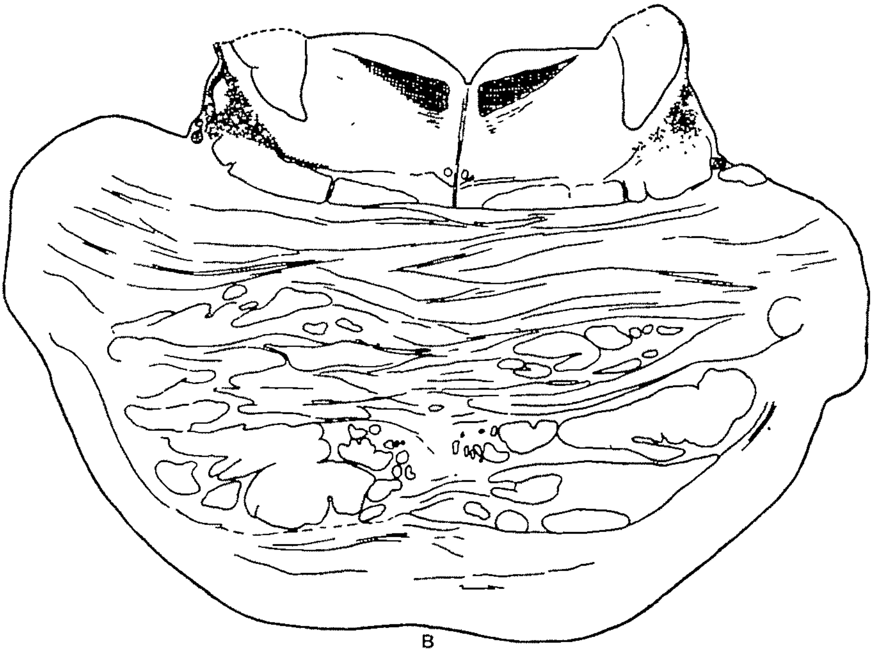
PLATE 1.

Ascending Degenerations.



A

Level of valve of Vieussens.

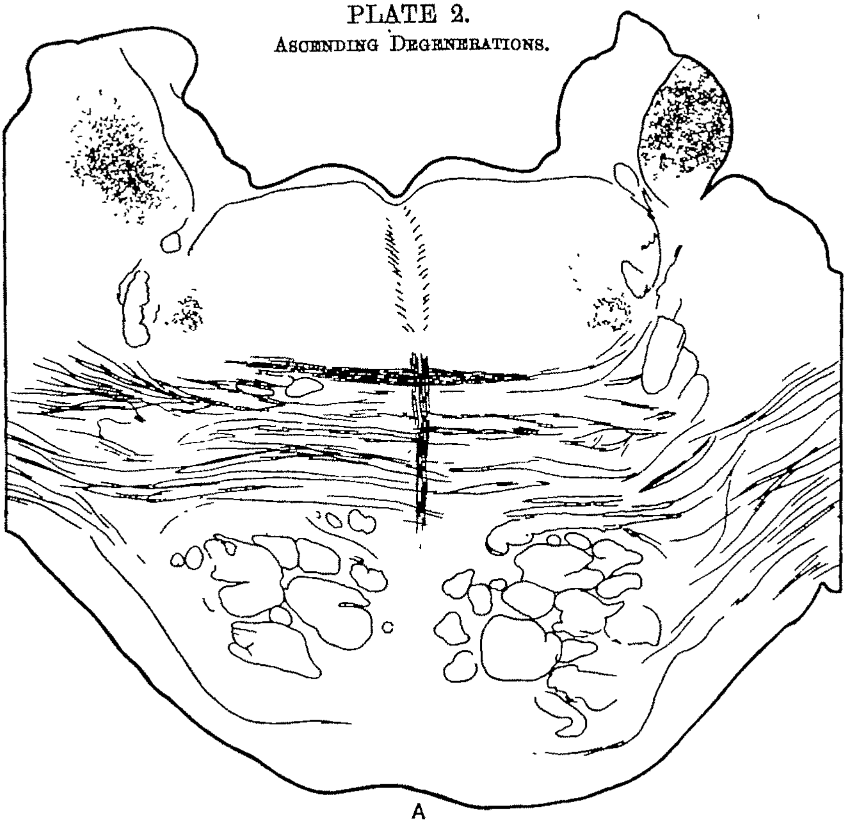


B

Upper part of pons.

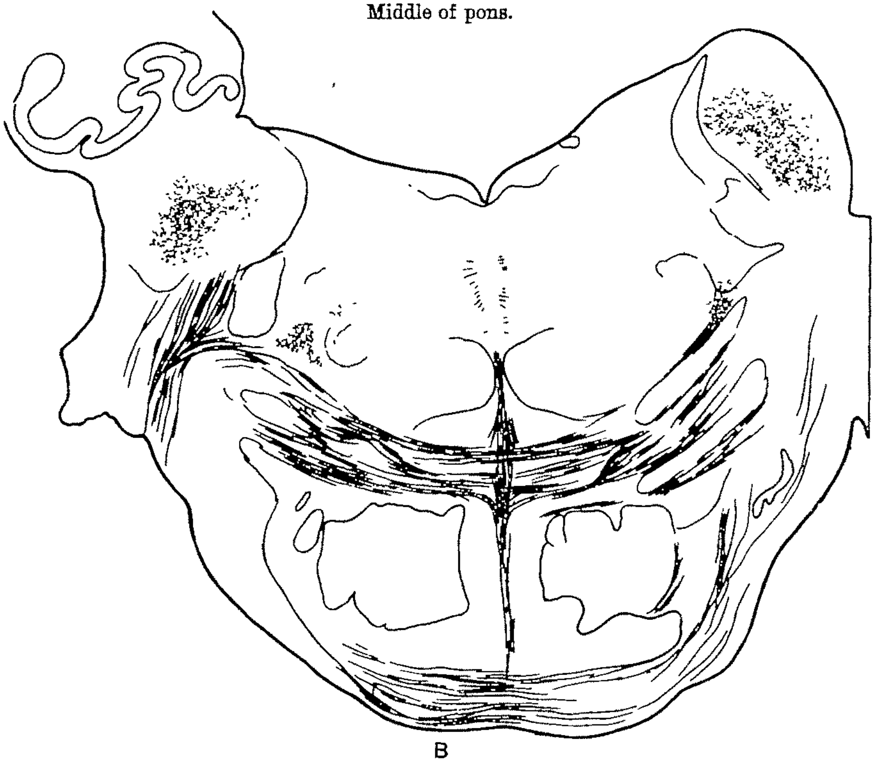
PLATE 2.

ASCENDING DEGENERATIONS.



A

Middle of pons.

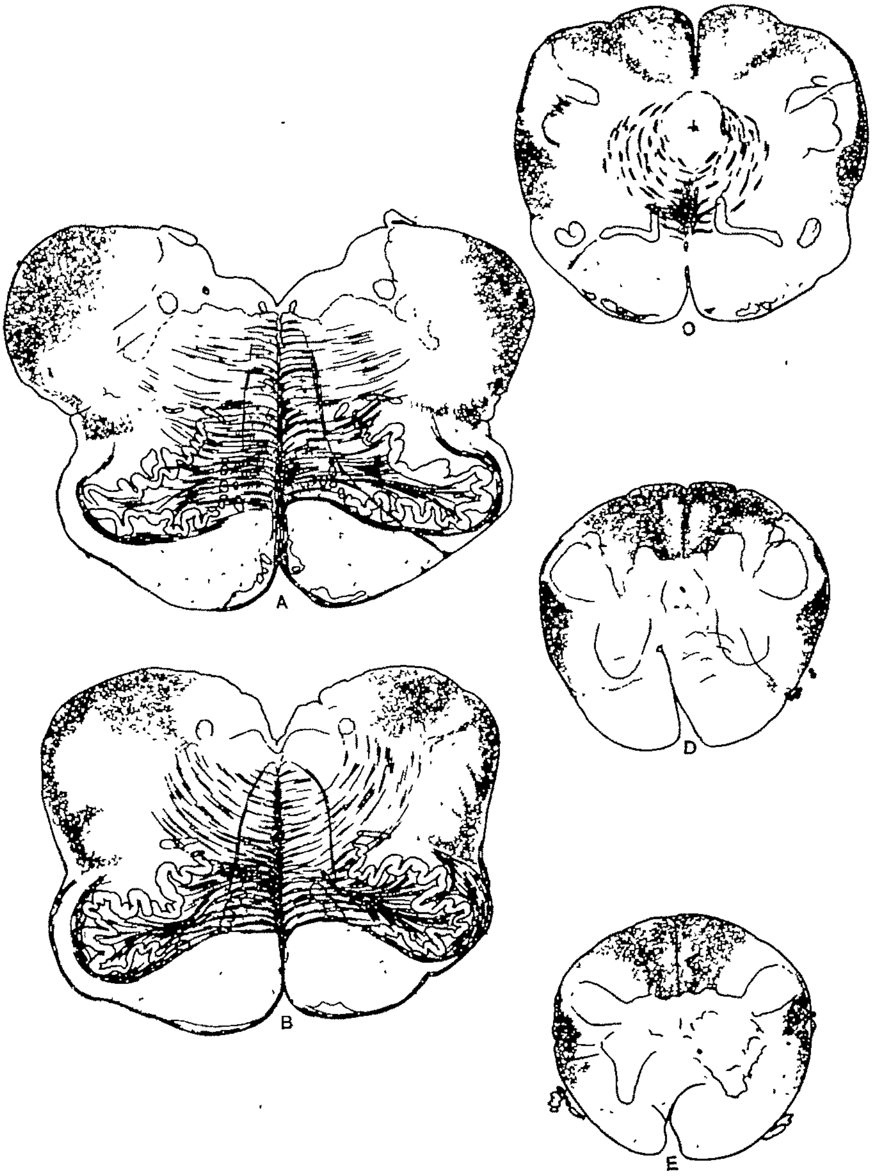


B

Level of facial nerve.

PLATE 3.

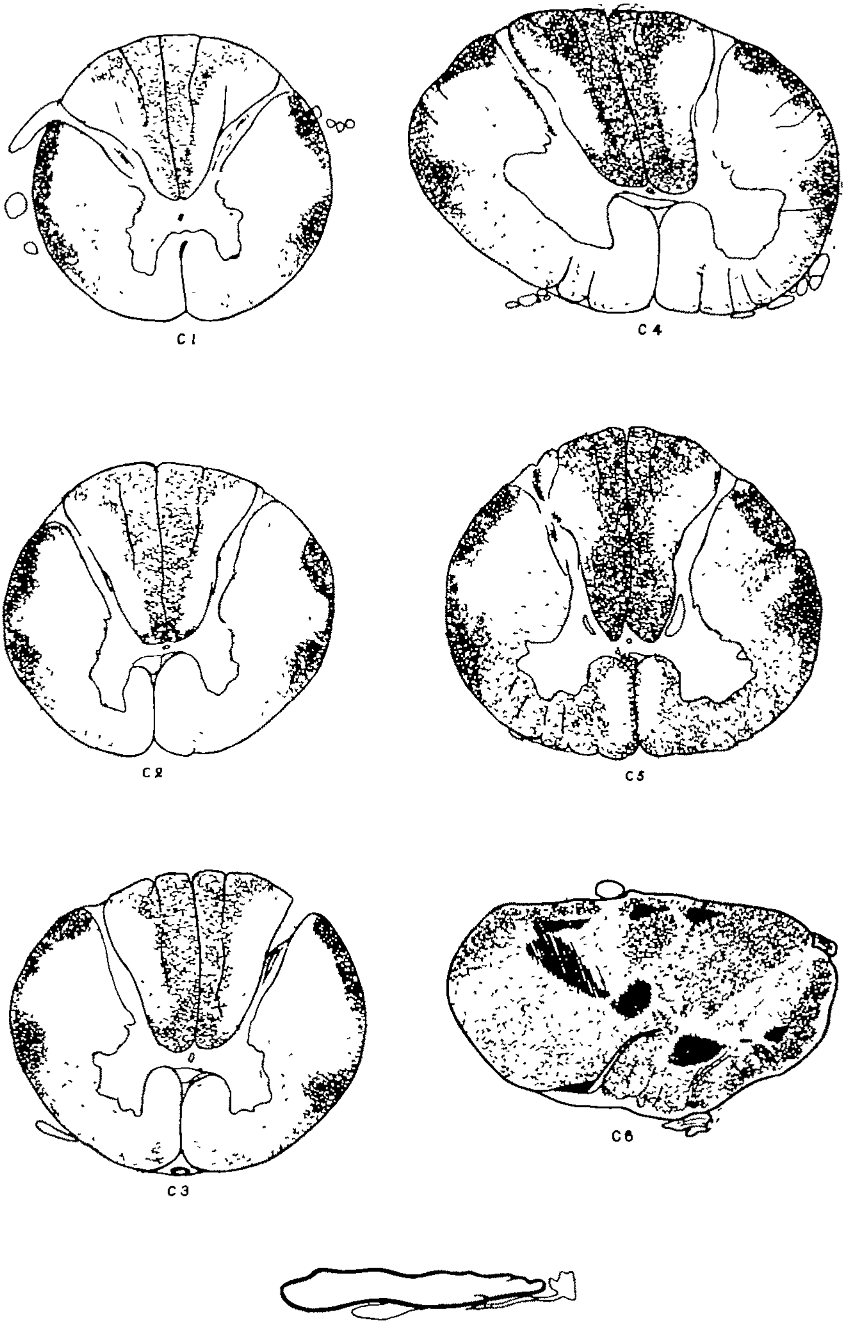
ASCENDING DEGENERATIONS.



A.—Medulla, upper. *B.*—Medulla, middle.
C.—Medulla, lower. *D.*—Pyramidal decussation, upper part.
E. –Pyramidal decussation, lower part.

PLATE 4.

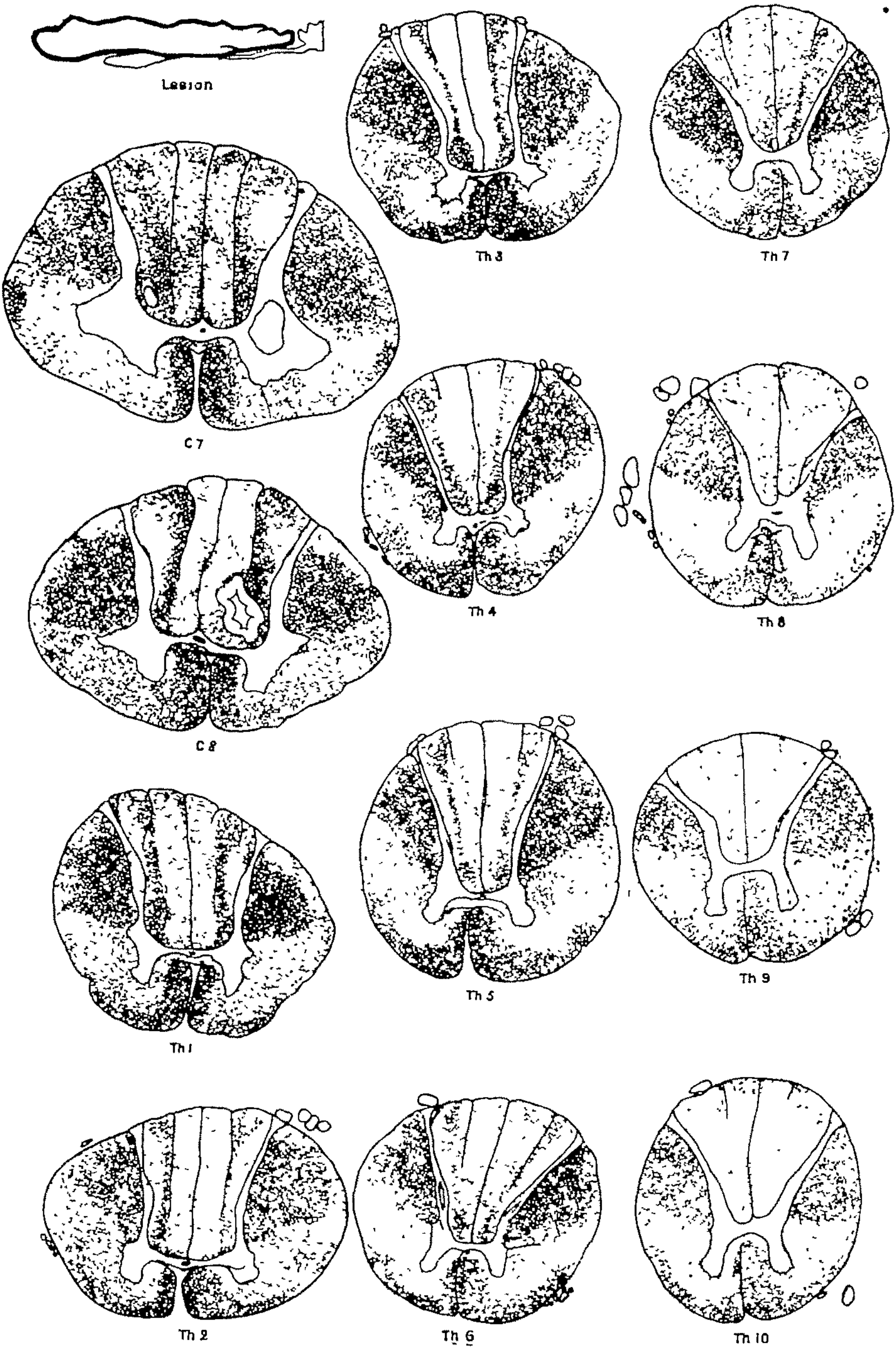
Ascending Degenerations.



Ascending degenerations in upper cervical cord.

PLATE 5.

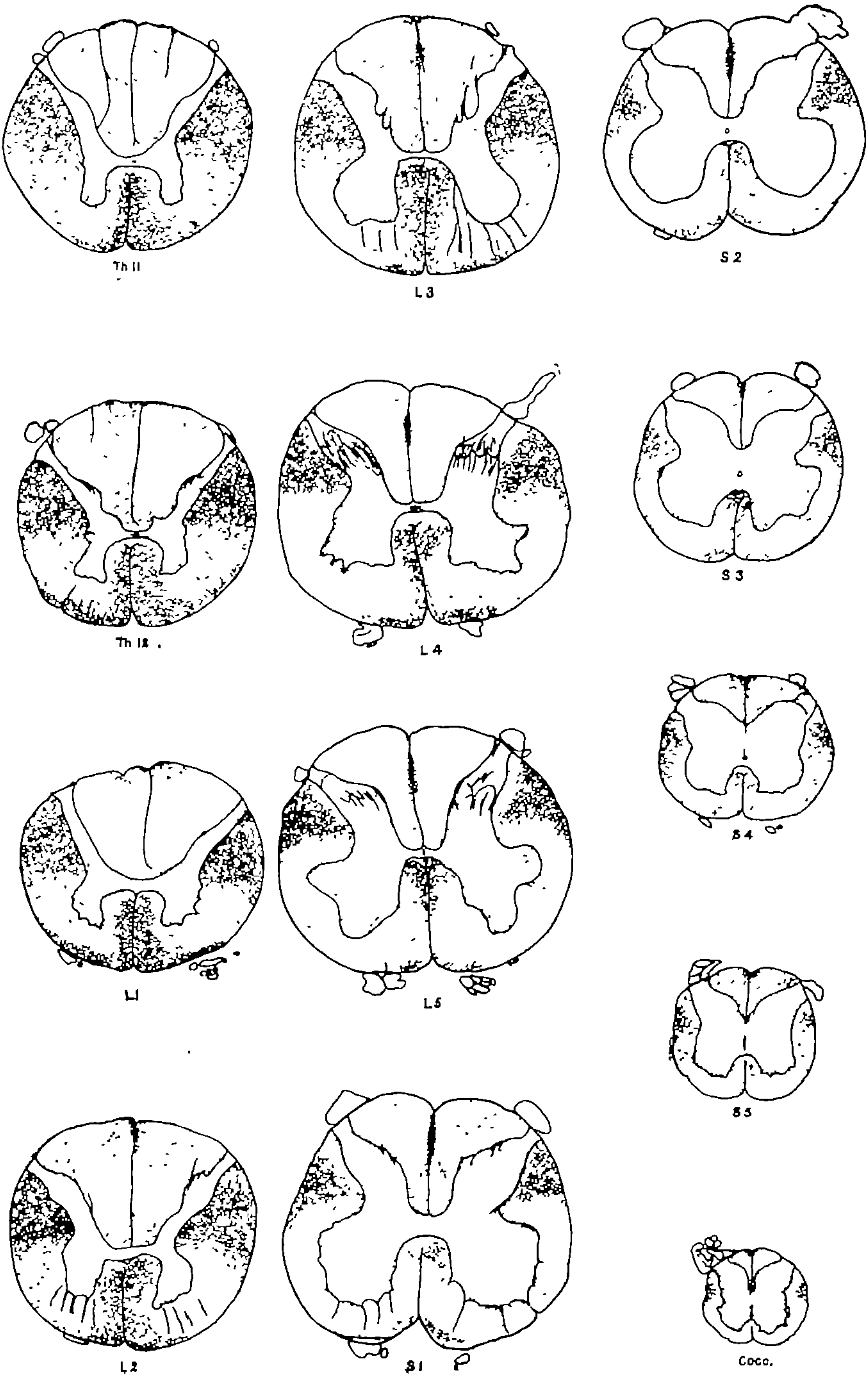
Descending Degenerations.



From level of lesion down to 10th thoracic segment.
"Tract X" in C. 7 and C 8 segments

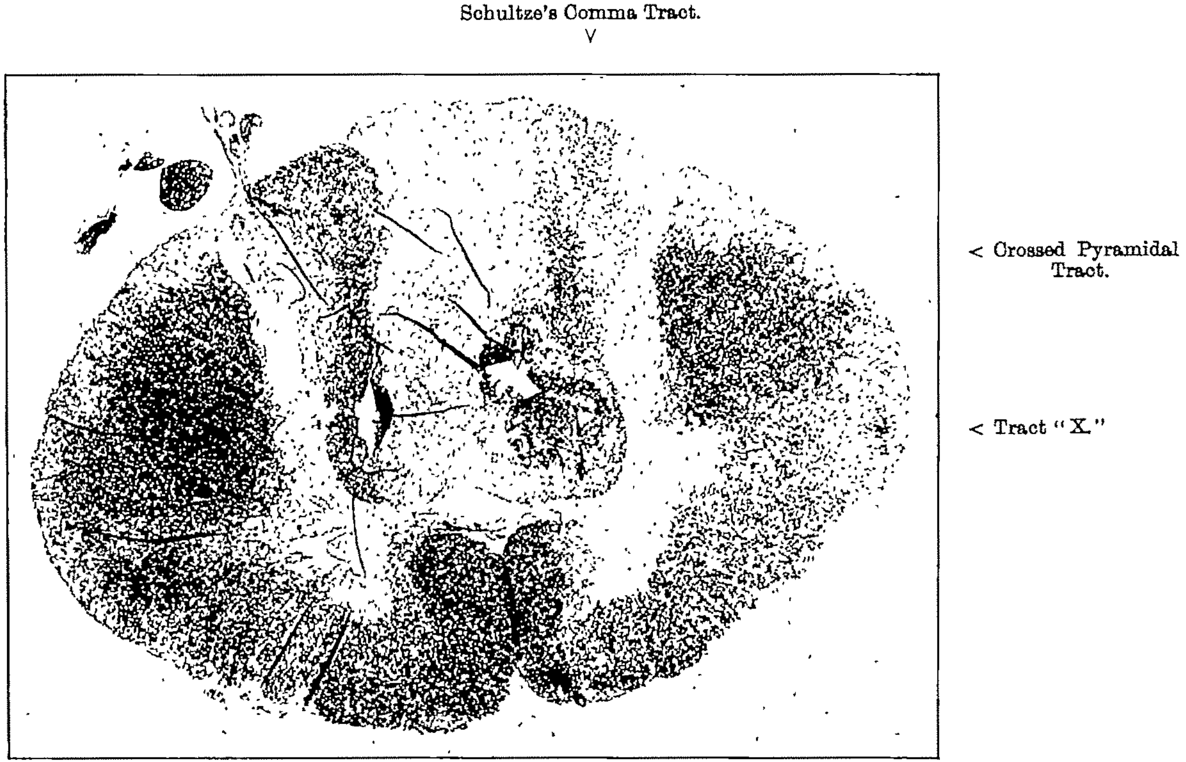
PLATE 6.

Descending Degenerations.

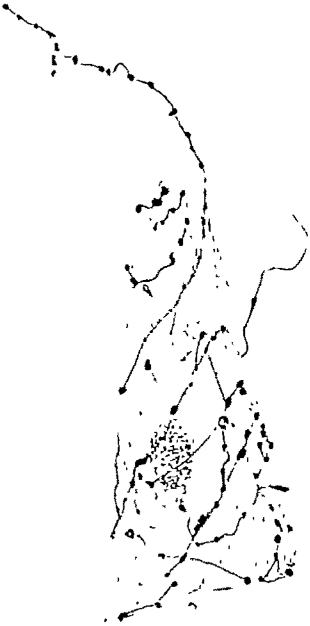


From 11th thoracic to coccygeal segment.

PLATE 7.



Photograph of section through lower part of 8th cervical segment



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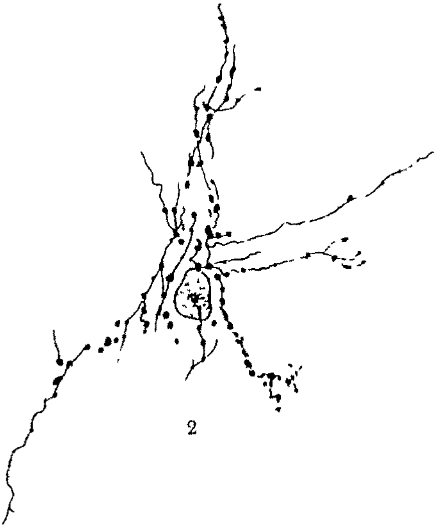

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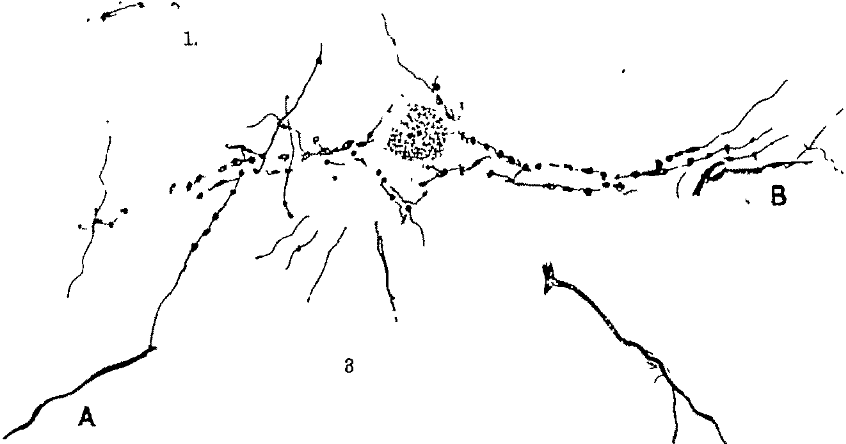

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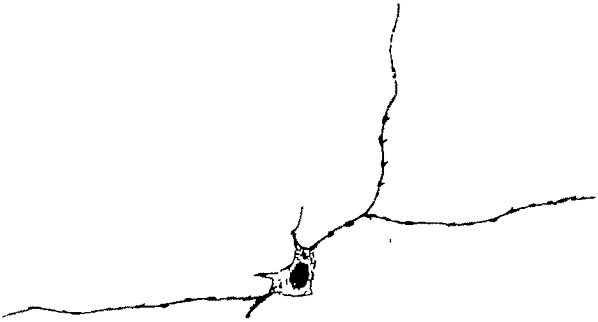

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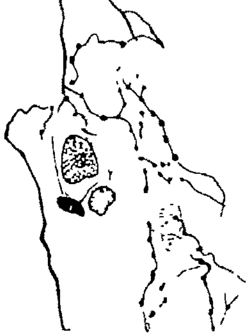

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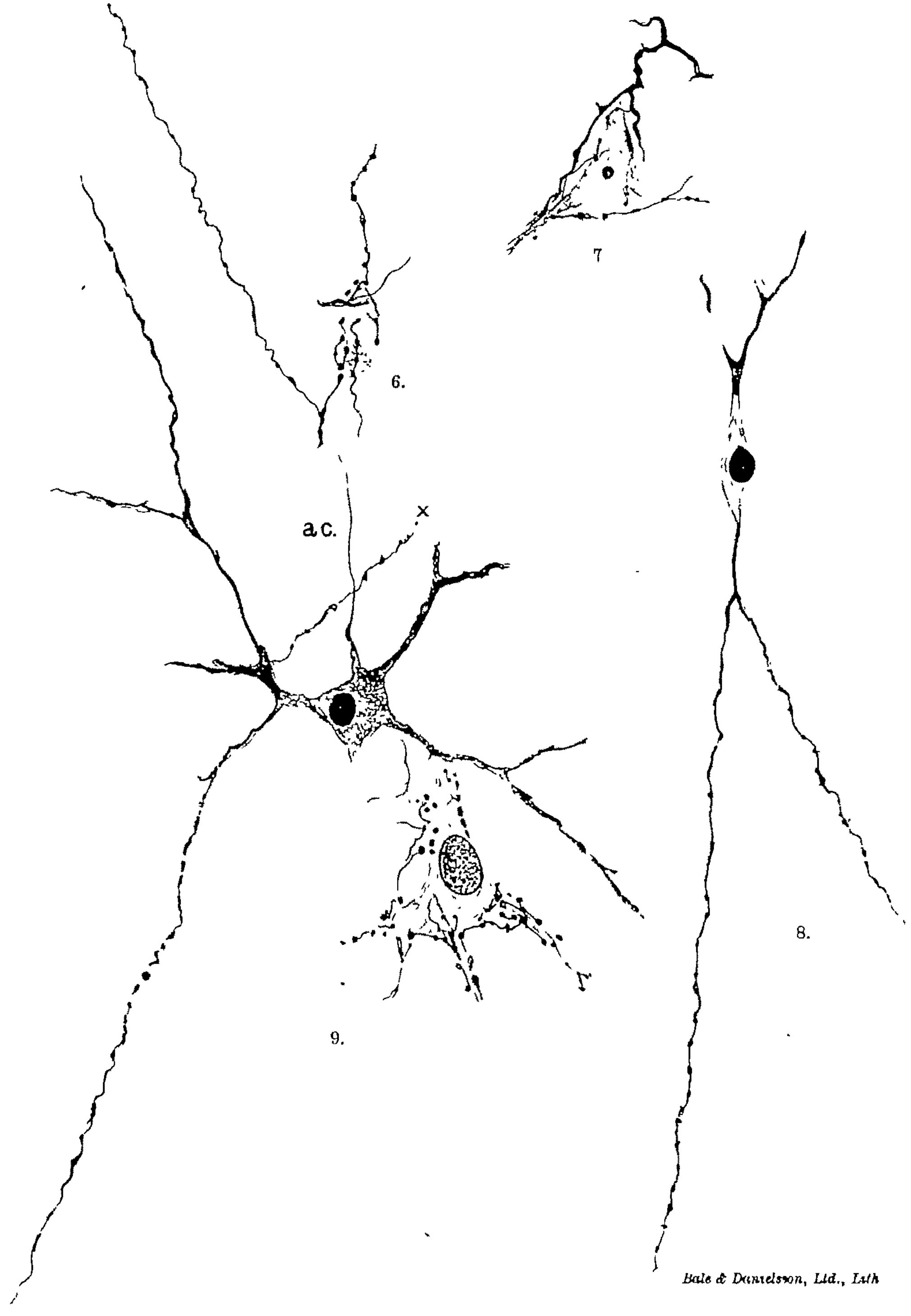

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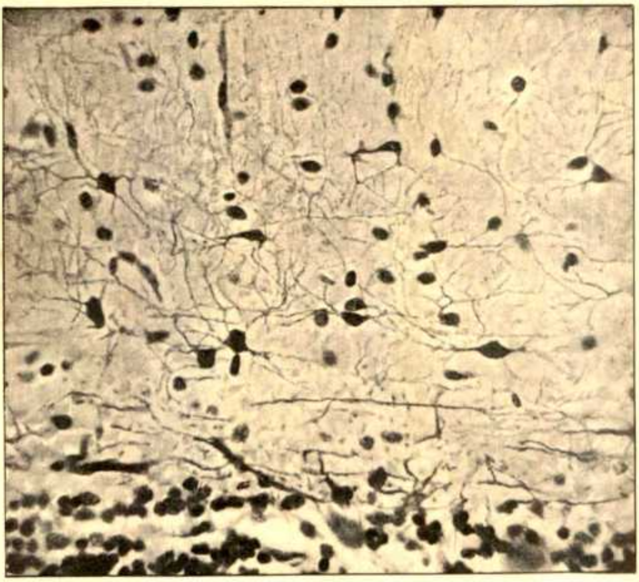

Fig. 10.

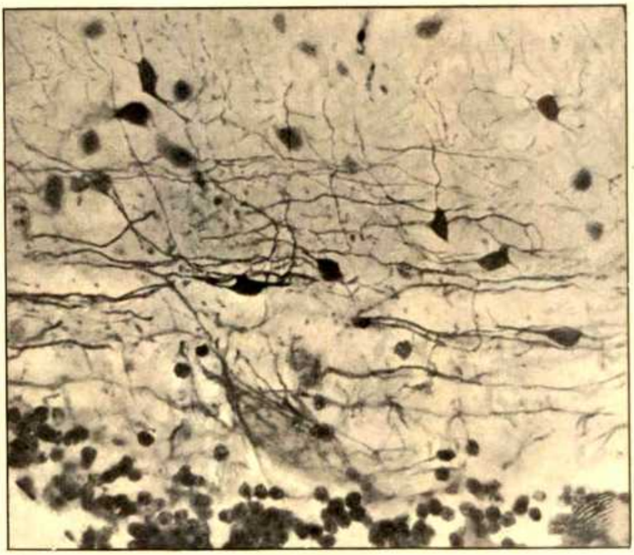

Fig. 11.

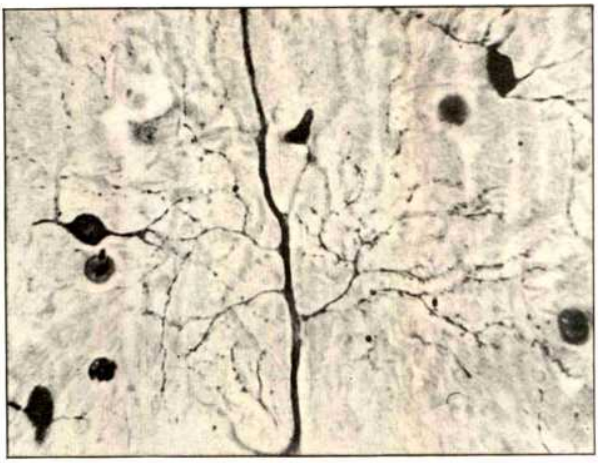

FIG. 12.

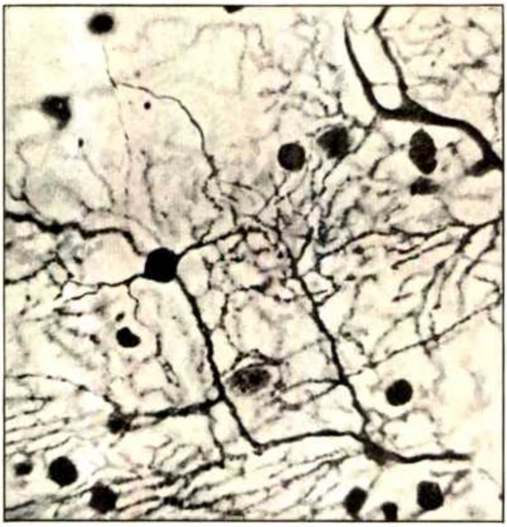

FIG. 13.

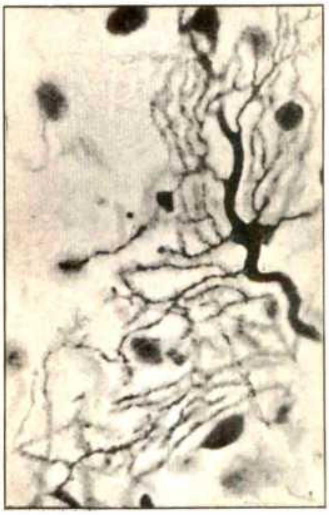

FIG. 14.

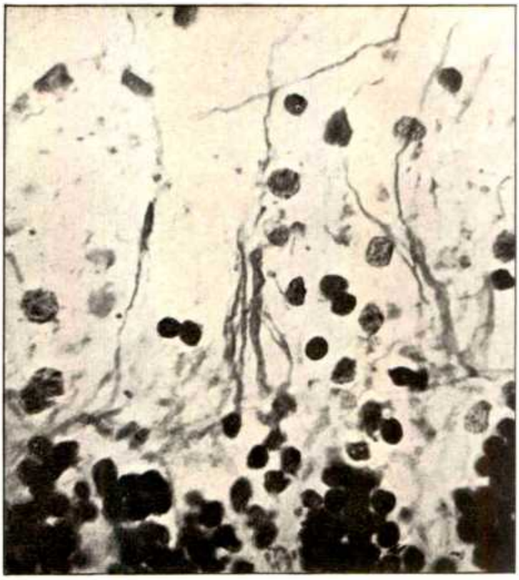

FIG. 15.

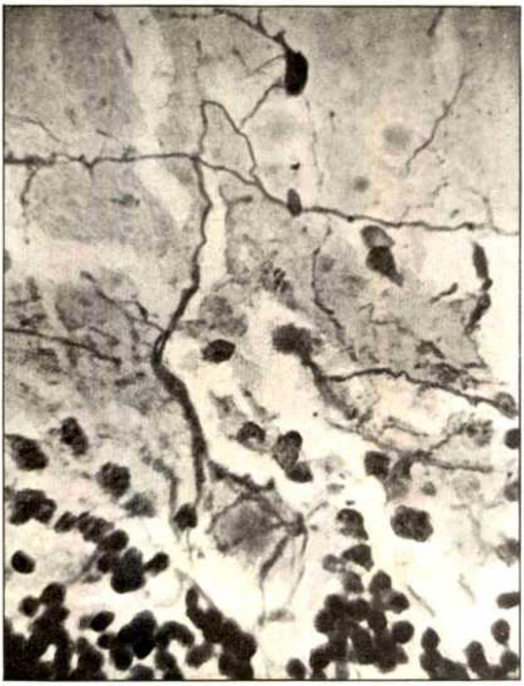

FIG. 16.

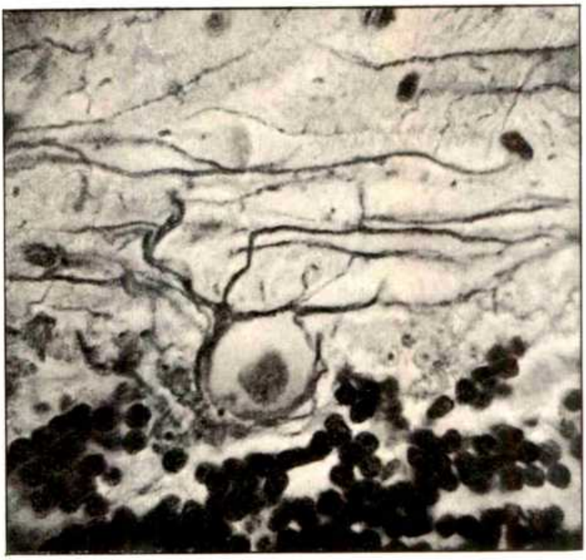

FIG. 17.

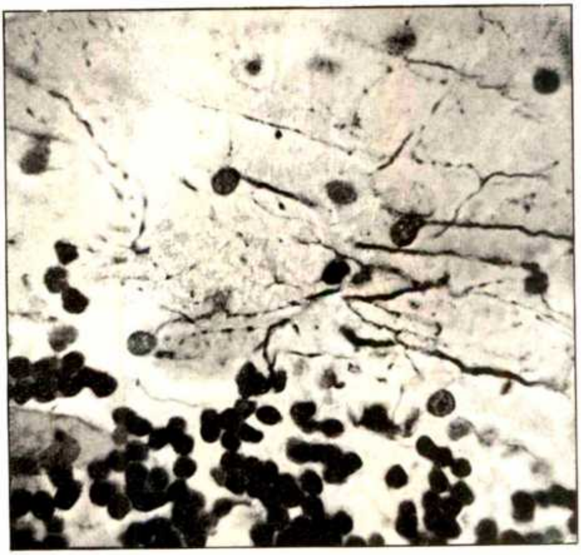

FIG. 18.

tract from which it is not sharply marked off, is seen at the level of the 5th cervical segment to be in apposition to the surface of the cord, whilst its anterior end or tail is spread out diffusely and does not cling so closely to the surface. It extends ventrally in amongst the anterior nerve-roots and its precise anterior limit cannot be defined owing to the existence of degenerated fibres in the ventral pyramidal tract (see later). On tracing Gowers' tract upwards through the medulla, it runs ventral to the dorso-lateral cerebellar tract, and when the latter enters the restiform body, Gowers' tract remains at the surface as a triangular mass with its base at the side of the medulla and its apex pointing inwards towards the posterior border of the inferior olive, where it becomes somewhat teased out. In the pons, at the level of the 5th nerve, the tract sinks inwards and lies internal to the trigeminus. It runs ventral to the 7th nerve and dorsal to the fibres of the middle peduncle, and sweeps outwards again to run upwards in the lateral fillet external to the superior cerebellar peduncle. In the lateral fillet its fibres are not scattered diffusely all over, but occupy the central part of that area, leaving a clear undegenerated space both to the outer and inner sides. From the lateral fillet its fibres can be traced to the valve of Vieussens, where the fibres of the tracts of the two sides are seen to intermingle. Sections through the posterior corpora quadrigemina show a few fibres in the region of the lateral fillet, diffusely scattered through the latter. The regions of the anterior corpora quadrigemina and the parts at a higher level were not obtained for examination.

So far as our results go, they are in accordance with those obtained by Hoche,[17] von Solder,[21] Patrick,[26] and Worotynski,[23] who trace Gowers' tract through the corpus trapezoideum to the region of the corpora quadrigemina. Its further course, whether, as stated by Mott, bending in a loop backwards to stream into the cerebellum, or, as Henneberg[27] maintains, sending a few of its fibres to end in the ventro-posterior part of the lateral nucleus of the optic thalamus, the main mass meantime turning back into the cerebellum, could not be studied in our case.

(3) *Pyramidal tracts, ventral and lateral.*—Although, in a section at the level of the 5th cervical segment, the ventral and lateral pyramidal tracts are sharply mapped out by their relatively scanty degeneration when contrasted with the marked degeneration of the remainder of the antero-lateral columns, yet on close examination they are found not to have escaped entirely. As we trace them to higher levels, distinctly degenerated fibres, although scanty and diffusely scattered, can still be detected throughout the pyramidal tracts in the cord, medulla and pons, *above* the level of the cervical lesion (see plate 3).

Whether this degeneration extends up to the motor cortex it is unfortunately impossible to state, since the structures above the pons are not available for examination. The question as to whether this degeneration is an "ascending" or a "descending" one is for the same reason, impossible of determination, for although on the one hand it might be that it is an "ascending" one, due to the spinal lesion, yet it is also conceivable that at the time of the accident a few of the cortical motor cells might have suffered, producing a descending degeneration in the pyramidal system. At the same time it should be remarked that there was no clinical evidence of such cortical injury. Even within a few hours of the accident, the patient showed neither mental impairment nor weakness of muscles above the level of the lesion.

CONCLUSIONS.

The following are the chief conclusions drawn from the study of this case:—

(1) There is a descending tract,—"Tract X," in the cervical region, situated ventro-external to the crossed pyramidal tract, separated from the latter by a distinct interval at a plane corresponding to the tip of the lateral horn of the gray matter. It does not extend below the level of the 8th cervical segment. It somewhat resembles in shape, but differs in position from the tract described by Helweg and v. Bechterew.

(2) The fibres of the lateral pyramidal tract, after a lesion in the cervical region, degenerate to the most caudal extremity of the cord.

(3) The ventral pyramidal tract extends as a distinct bundle to the 4th sacral segment, below which level its fibres become diffused throughout the anterior columns and are traceable to the most caudal extremity of the cord.

(4) The efferent ventro-lateral tract extends downwards as far as the 4th sacral segment. Below this level its fibres become diffused over the antero-lateral column. Those fibres which lie nearest to the anterior median fissure extend as a distinct tract farther caudally than the outer fibres.

(5) Degenerated fibres occur in the white commissure for three segments below a lesion in the lower cervical region.

(6) Descending degeneration in the posterior columns occurs in the "comma" tract of Schultze as far down as the 9th thoracic segment. Below that level, degeneration continues successively in the "septo-marginal" tract of Bruce and Muir (10th thoracic to 2nd lumbar), in the "oval field" of Flechsig (3rd lumbar to 2nd sacral) and in the "triangle médian" of Gombault and Phillippe (3rd sacral to caudal extremity). No demarcation can be traced between the fibres of the "comma" tract of Schultze and those in the other three descending tracts farther down in the posterior columns. It is therefore probable that they all form one long combined system.

(7) Ascending degeneration of the posterior columns above the level of a cervical lesion does not stop abruptly at the posterior column nuclei. Some degenerated fibres run on both in the internal arcuate and in the postero-external arcuate paths.

(8) The ascending antero-lateral tract of Gowers extends upwards through the lateral fillet through the superior medullary velum (where it mingles with the fibres of the opposite side) to the posterior corpora quadrigemina. In the lateral fillet, the fibres of Gowers' tract occupy the central part of that structure and do not extend to the margins, either externally or internally.

(9) Degeneration also occurs in the pyramidal tracts in

the cord, medulla and pons above the level of the cervical lesion. Whether the degeneration be a true ascending one or not, it is impossible to determine from this case.

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OBSERVATIONS ON THE MINUTE STRUCTURE OF THE CORTEX OF THE BRAIN AS REVEALED BY THE METHYLENE BLUE AND PEROXIDE OF HYDROGEN METHOD OF STAINING THE TISSUE DIRECT ON ITS REMOVAL FROM THE BODY.

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I.—Concerning the Technique of the Method.

Since the preliminary notice of this process, which appeared in the Autumn number of Brain, 1900, I have been engaged in making further trials with it, from the results of which it appears as though the reaction is even more capricious and uncertain than I then thought, and that for its success other conditions must be complied with beyond those specifically mentioned in my first account.

A series of trials, closely following those, the results of which I published, on a dozen or more different brains, nearly all resulted in complete failure, no trace of the reaction being obtained except once in the case of a cerebellum, and this was very good.

During this time the weather had been dull, and the bottles containing the tissue to be stained had been placed far from the window of the room they were in.

As nearly all the successes happened during the summer months, it occurred to me that perhaps heat or light had something to do with the matter.

So far although not able altogether to eliminate the effects of heat in aiding the reaction, I think from the trials I have made that at any rate it takes a very subordinate part; but as regards light this seems to be essential. After many failures, using different proportions of peroxide of hydrogen, different solutions of methylene blue, and keeping the tissue at different temperatures I have obtained a success or modified success in a certain number of pieces which have been kept fully exposed to the light, and perhaps the modified nature of the success is to be ascribed to the season of year, and the extremely dull weather that has been the rule during the time these trials were made.

In November, December and January, during which period these experiments took place, the active properties of light is at its lowest, and very likely as this increases in the summer months the results will be found to get correspondingly better.

The series of trials to show the effect of light were made on pieces of tissue from twenty-four brains; and they were put into a mixture of a 1 per cent. solution of methylene blue—four parts, a 10 per cent. solution of peroxide of hydrogen—one part, and left for nine to eleven days against a window and not interfered with. Of these fourteen turned out more or less successfully, but only five or six were really good, and most of these were pieces of cerebullum.

That very cold weather has a deterrent effect on the process is rendered probable by the fact that during the late cold weather in February and March not a single success was obtained in nearly thirty specimens of different brains, but later on as the weather became warmer, using the same solution and under similar conditions, successes were again the rule.

One other point must be noted—I have never got the reaction in the brains of animals freshly killed or human brains very shortly (a few hours) after death. It seems that a certain stage of decomposition in the tissue is necessary before it takes on the stain in the required way.

As lactic acid is produced during this decomposition I have lately added four to eight drops to a pint of the staining solution, but although some good results have been obtained after this addition, it is yet too early to speak definitely as to the desirability of this addition as a routine practice.

Further trials since the above was written seem to confirm the idea that the addition of lactic acid is desirable. I even add as much as three drops to 40 cc. of the staining mixture (the amount usually employed in individual cases). This addition renders the tissue more brittle—but in cases where pieces of tissue were placed in this strongly acid mixture and others from the same brain in a very faintly acid solution, whilst the former were successful the latter were not, other conditions being the same.

In the previous notice of the method, reference was made to the partial character of the reaction, and it was mentioned that only little patches of the tissue show it, and the first layer and superficial part of the second never stain successfully. To this must be added the fact that in the cerebellum with very few exceptions the reaction occurs at the bottom of a sulcus and usually gets less marked towards the top of the sulcus, and although the granules are deeply stained, yet in this layer there is never any sign of the reaction. No appearance of the processes of the granule, cells, &c.

It is also in the deeper or interior parts of the block of tissue that as a rule the best sections are got, sometimes indeed when the first few outside sections of a block show scarcely any reaction, those more in the centre will show it well.

In passing I may remark, as it may have some bearing on the question as to the nature of the small darkly-stained structures in the cortex, whether they are of nervous or neuroglial origin, that the latter form of cell in the medullary substance of the cortex are often seen, both in the cerebrum and cerebellum, but they stain of different colour to the nerve-cells and processes. Both the cell body and processes are of a somewhat pale reddish tinge.[1]

In the places where the re-action occurs fully the process probably brings out greater detail of structure than any hitherto known method, for it is quite equal to gold, silver or mercury in picking out the fine ramifications of nerve fibres, but further than these it appears to differentiate between different nerve-cells, so that while the pyramidal system and the Purkinge cells are stained faintly others are very darkly stained. The latter, which are further distinguished by size and shape, would appear as though in ordinary methylene blue staining they are not shown at all or probably only their nucleus is stained, for we see in unsuccessful preparations of cortex stained by the peroxide method that there is no trace of them to be found, whilst in slightly successful pieces one of the first indications of the reaction is the picking out more or less clearly of these cells.

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[1] Occasionally the neuroglial fibres of the first layer in pathological conditions stain clearly as dark, smooth, wavy, or crenated fibres. A similar appearance is sometimes seen in the cortex cerebelli when the radial or Bergmann's fibres stain as above noted, and appear very similar to the basket fibres.

While the opaque character of the impregnation with silver or mercury more or less obscures all detail in and immediately around the cell, the present method, even in the case of the darkly-stained cells, allows fully the interior and surrounding detail to be seen.

And finally, another advantage of the method is that it appears to be permanent, for some of the sections I have now before me have been kept for over two years and show no deterioration.

The foregoing remarks regarding the technique of the method must be taken as representing conclusions only provisionally arrived at, and one reason for publishing these observations in what may be regarded as a premature state is that others may be induced to experiment with the complicated conditions necessary for success in this very elusive process, a process, however, which gives promise of being of considerable service to those engaged in the study of the structure of the nervous system in health and possibly also in disease.

Another reason is that as the specimens I have show clearly the appearances on which my deductions are founded, I think it advisable to publish the results without waiting for further developments in the method; for although as mentioned before, so far the sections appear permanent it is yet possible they may deteriorate in process of time, or get damaged, and with the uncertainties of the method in view, I may not be able to replace them by others as good.

The following summarises the steps in the process as at present adopted:—

(1) An old-standing 1 per cent. solution of methylene blue (Grubler's pat. b. x), kept exposed to light and air till its *pithy* smell has disappeared. Probably the addition of four to eight drops of lactic acid to the pint is an advantage.

(2) A mixture of four parts of this to one of a 10 per cent. solution of peroxide of hydrogen. The pieces of tissue are placed in this mixture in a fairly capacious bottle, and kept exposed to light, and if very cold weather at a temperature of 25° to 30° C. for eight or nine days.

(3) Twenty-four hours or more in 10 per cent. molybdate of ammonia.

(4) Washed in running water about twelve hours (all day).

(5) The following evening and night in several changes of absolute alcohol.

(6) The next morning through several changes of xylol for two hours.

(7) Two hours in paraffin bath.

Following these instructions, in the summer time under favourable conditions of light and temperature, the successes will probably be in the proportion of nine or ten to one failure; whilst during the winter and dull weather the reverse will be the case—only one success to about nine or ten failures.

In the following pages I propose to consider briefly the structures shown by this method in the cortex of the cerebrum and cerebellum, and to suggest what seems to me to be the most probable inferences to be drawn therefrom. In the description given of the molecular layer of the cerebellum I am quite aware that the main features noticed will be familiar to workers in Golgi's method; but even so, they appear worthy to note, inasmuch as this latter method has, I believe, been chiefly limited in its application to the cerebellum of animals or human embryos, and therefore, at the least, the results I get are a corroboration of those obtained by Golgi's method; especially as it has been contended that the so-called staining of this latter process is due to impregnation about the nervous structures of precipitated metal in a fine state and not to a true staining of these structures.

Of course the limitation of the stain, in my method, to the molecular and Purkinge cell layers is a serious drawback to its utility in tracing the further course of nerve-fibres.

II.—CEREBRUM.

This method reveals around the pyramidal and giant-cells of the cortex a delicate beaded network (see figs. 1, 2, and 3) loosely enveloping the cell-body and its apex and processes, and extending along the latter as far as it is ever possible to trace them in sections. In some specimens the beaded network can be clearly seen enveloping like an open-work stocking the dendrites of the giant-cells for a distance of 300 μ. The beads on this network stain, as do the fibres, nearly black, and vary considerably in size and shape, some being round, others of irregular contour, and others ring-like. The large irregular beads sometimes appear as if two or more small ones had blended together. The usual size of these beads is about 1 μ, but large ones are met with of 2 or 3 μ in diameter. Where the branching of the network occurs, the fibres always appear to spring from a bead, and not from intermediate portions of the fibril. The distance apart of the beads varies considerably; over some of the giant-cells, where they are generally large in size, they may be clustered very closely together, but a distance of several μ usually separates them. Now, this network does not appear as a closed network drawn over the cell; but presents on all sides tags formed by beaded fibres, passing off from it into the surrounding matrix (see figs. 2 and 3), and it is not unfrequently possible to trace some of these fibrils into the stouter fibres, which have all the appearance of being dendrites from neighbouring cells;

also, in like manner, branches can be found which give off fibrils to supply the network of two adjacent pyramidal cells (see fig. 5).

I wish to emphasise the statement that fibres coming off from different parts of the network can be traced running in different directions in the surrounding matrix, and in some cases can be traced to the thicker branches, evidently proceeding from different cells, because it implies that the cells from the branches of which the network is ultimately formed are in organic continuity one with another by means of this network, and this holds good whether, as some think, the network is an extension of the collaterals of other cells, or whether, as I think, it is an extension of the dendrites of the small dark cells. This beaded network is apparently a very persistent structure, and is still found in brains which are the seat of extensive pathological changes, *e.g.*, in the brains of general paralysis and dements.

Two varieties of cells are distinguished by this method—pale and dark. A cursory reference was made in my former communication to this fact, and it was pointed out that whereas the cytoplasm of the majority of the cells (pyramidal and giants) stains very lightly others are picked out which stain very darkly.

The cytoplasm of the pyramidal and giant cells stains uniformly and very lightly, so that the apex and dendrites are not prominent features and can only be discovered near the cell body. The nucleus is plump and consists of darkly stained granules closely packed and contained in a definite and darkly stained membrane. The nucleolus as a rule is paler than the nucleus and indistinctly outlined.

It will be seen that this description applies also to pyramidal cells stained by the intra-vitam method.

Amongst the pyramidal cells we meet with instances of dark staining, more especially among the giants, but the infrequency with which these occur in proportion to the numbers which are lightly stained points to a pathological cause for this difference.

And furthermore, one always meets with more dark-stained pyramidal cells in brains which have undergone obvious pathological changes than in brains from cases of recent insanity.

Although as just stated in the pale-stained pyramidal cells the dendrites are a very insignificant feature and can rarely be traced far, yet in the brains of certain dements of long standing we find many of the pyramidal and giant cells very deeply stained and showing a great wealth of dendrites which are irregular in contour and can be traced for long distances. Now these processes, both apices and dendrites, appear to give off nearly at right angles numerous very fine fibrils.

Even although we grant that this dark coloration of the cell is pathological, this does not alter the significance of the fact in showing the extensive ramifications of the cell branches, for it will scarcely be admitted that a morbid process will have any marked effect in the production of this extensive ramification, or in causing any decided change in its general configuration. We must admit, I think, that the processes thus shown are normally present although only occasionally stained.

One point must be borne in mind, when the cell and its processes are deeply stained the fine investing network is naturally obscured, and I think that quite possibly the irregularity noticed in the dendrites is due to this investing network and that some at least of the fine fibrils mentioned as passing off from these dendrites are in reality fibrils *coming to* them to join the network.

In deeply-stained giant-cells sometimes the axis cylinder can be seen passing off as a darkly-stained, perfectly smoothly outlined fibre, but which, after passing 50 or 60 μ from the cell, loses its colour and appears as a smooth, very pale strand; and these stout pale strands representing axis cylinders are generally seen running in the lower part of the cortex.

The dark cells of the cerebrum present considerable diversity in shape and size, but the great majority are between 20 and 24 μ long and of a more or less spindle shape, although not unfrequently one meets with square or pentagonal forms (see figs. 4, 8, 9).

While the cytoplasm and its extensions stain deep blue, the nucleus appears as a homogenous and black mass. The processes of these cells, which are given off all around the body, spring from it at once as comparatively slender branches which can often be traced for 200 μ or more. They do not branch much as a rule (I am not referring now to the very delicate threads given off all along nearly at right angles to the main stems), and generally begin as shaggy fibres, passing, as they get finer, into moniliform or beaded fibrils.

It is from the dendrites of these cells that I believe I have been able in several instances to trace directly the beaded fibrils of the network about the pyramidal cells.

Apart from the shaggy processes above referred to, sometimes one sees a perfectly smoothly contoured fibre of small calibre passing off from the cell, but in all cases where I have seen this smooth fibre it could only be traced a short distance before passing out of the plane of the section, or perhaps (as with the axis cylinder of the great cells) losing its colour. As very often this smooth fibre is given off from the upper part of the cell and runs towards the surface of the section, it seems probable that some, at any rate, of these dark cells are Martinotti's cells (see fig. 9).

These dark cells are found scattered throughout the cortex from the second layer inclusive down to the medullary substance.

These dark cells are chiefly if not entirely limited to the region of the granule cells in the occipital cortex so far as my observations show. And they are clustered together in amongst the granules in greater number than I have met with them in either the frontal or ascending frontal cortex.

There is never any sign of a beaded network around them or their processes.

Briefly summarised, the following are the main features shown by this method :—

(1) That there are certain cells, the body and dendrites of which stain very lightly and their nucleus dark, granular, and sharply defined, and that the axis cylinder of these cells, when it can be seen, is smooth and of a very pale blue colour.

(2) That a network of fine beaded fibrils loosely invests these cells and their protoplasmic processes, and that beaded fibrils pass from this network at many different points, and that in some cases these can be traced joining to stouter, somewhat shaggy fibres.

(3) That there are certain darkly stained cells, generally small, often pear-shaped or pentagonal, and which give off fine dark-stained processes, which processes can often be followed for very long distances with very little branching.

The finest branches of these cells are beaded or moniliform, and appear very similar to the beaded fibrils of the network.

(4) These dark cells show no signs of any network around them.

Sometimes fibrils can be directly traced from these cells to the network. (This last observation must be separated from the former because before it can be accepted as a fact it will require further corroboration.)

The above, I submit, are four observations that can be verified by any one inspecting my specimens or preparing others by the same method, and from these I would draw the following inferences: First, as to the origin and significance of the beaded network. There are good reasons against the supposition that it is a supporting network of neuroglial fibres, for in the first place the neuroglial elements do not usually stain by this method, and when they do they stain of a rather pale reddish tint and their fibrils are not so distinct or so dark,[1] and in the second place Golgi's method shows in the ending of certain peripheral sensory nerve fibrils just such a beaded appearance as is seen in the network, and no process that I know of has ever shown anything like a beaded appearance of the neuroglial fibres.

Excluding then the neuroglia I think we may fairly certainly assume that the network is formed by the processes of nerve-cells.

As to their being the extension of the collaterals of axis cylinders we must bear in mind that the axis cylinders, as seen in the lower part of the cortex by this method, appear as very pale blue and smoothly contoured fibres, and collaterals are not certainly stained at all. By this I mean I have never seen a dark collateral given off from one of the pale axis cylinders, although of course it is possible that the ultimate terminations of the collaterals may stain differently from the same fibre at its origin from the axis cylinder.

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[1] *See* foot-note, page 240, for exception to this statement.

As to its being an extension from the dendrites of the pyramidal cells, these usually are very pale and soon disappear from the plane of the section, and although they sometimes colour deeply and can be traced for long distances, it is not only in these cases, as we should expect if they gave origin to the network that this latter shows, but even when the pyramids and processes do not colour at all.

Now in favour of the assumption that the network is formed by extension of the dark cells we have the fact that the processes of these cells always stain deeply and of the same colour as the network, and in all physical characters, they appear similar to the fibrils of the network.

And finally, I believe that in several cases I have been able to trace a fine beaded fibril coming from a dark cell directly to a network, so that the balance of evidence seems to be in favour of this network being as it were an extension of the ultimate fibrils proceeding from the small dark cells.

If we accept the above interpretation it must lead to considerable alteration in our conception of the arrangement of the nervous elements in the cortex of the brain, *for it implies that there are at least two systems of cells present, the pale or pyramidal variety, and the small darkly-stained variety, and these latter branch in all directions and their dendrites ramify and ultimately form a network about the former, and the whole system of dark cells is in absolute continuity one with another through the medium of the network.*

III.—CEREBELLUM.

Figs. 9 and 10 give a general idea of the appearance of the cerebellum as shown by this method.

In fig. 9 chiefly the small cortical cells (Shaffer's cells) are shown, and although numerous delicate fibrils are seen given off from them and interlacing in all directions, yet the photograph gives but a very poor idea of the wealth of fibrils actually shown in the sections, and to see which it is necessary to shift the focus.

In fig. 10, more highly magnified (×400) the lower half of the cortex only is shown, and the dark cells seen are chiefly basket cells. In the lower border of the figure there is a Purkinge cell enveloped in its sheath of basket fibres.

The Purkinge cells, as a rule, stain lightly, and show no trace of chromophilic material; the nucleus is dark and granular, and with a sharply defined border; the nucleolus varies, but is often pale and indistinct.

Variations in this way of staining have been pretty frequently met with, but as I am dealing with pathological material, it seems probable that these variations are due to morbid conditions, and I have taken as normal the form most frequently met with in the young and in recent cases of insanity, and which is most nearly allied to the staining obtained by the *intra-vitam* method. In the majority of cases the branches of the Purkinge cells either do not stain or else stain very lightly, but occasionally these also are dark, and indeed sometimes darkly coloured branches are seen originating from a cell with pale body. Of course it is only when they are darkly stained that we get a view of the great wealth of branches given off and showing then as a rule, little lateral projections or thorns (figs. 12, 13, 14).

Even if the supposition is correct that a dark colour implies a pathological condition of the cell and processes, this does not necessarily mean that thorns are a pathological feature, but that under normal conditions they do not take on the stain.

The ramifications of the Purkinge cells extend quite up to the surface of the layer, and the main stems do not bear thorns, only the terminal twigs.

These thorns take the shape of a short delicate lateral fibril ending in a knob. I find that they are of common occurrence when the branches from which they spring stain deeply, and when met with they occur all along the processes, and not one here and there, although Obersteiner says ("Anatomy of Central Nervous Organs" translated by Hill, edition 1900, p. 405), speaking of Golgi's method:—"In perfectly successful preparations of the brains of adults no such excrescences appear, only a granule here and there is seen attached to the processes of an isolated cell."

In the case of a man 68 years old, suffering from melancholia, although there was very manifest atrophy of the branching fibres of the Purkinge cells, yet the lateral projections with knobbed ends were very distinctly shown (fig. 12). The atrophy referred to is not a necessary accompaniment of age, as in another man of 68, the branches were as plump and broad as usual.

In the lower half of the molecular layer in sagittal sections (across the direction of the foliœ) are seen a number of stout (1 to 1·5 μ), smoothly contoured, wavy or crinkled fibres, which run for the most part parallel one to another and to the surface of the layer and stain deeply. Not unfrequently one of these fibres bends back abruptly and runs in an opposite direction.

These fibres originate (in part at least) from small (12 to 15 μ), oval or pear-shaped cells lying in amongst them and usually with their long diameter in the direction of the fibres. These cells stain deeply, and for this reason it is often difficult to distinguish the nucleus from the cell body. Besides the stout, smooth, parallel fibre, which is said to be the axis cylinder and which passes generally from one end of the cell (and sometimes apparently one from each end), other branches are given off. These generally divide near the cell into very delicate fibrils, which may appear to end shortly, as for example when they run in the direction of the granular layer, or else as when they spring from the upper side, may often be followed for very long distances without further branching. Some of these long, fine branches running upwards can be traced to within a few microns of the surface of the layer and there double back and run down in an opposite direction, traversing in their downward course considerably more than half the width of the layer, so that altogether these delicate processes can sometimes be distinctly traced for the surprising distance of over 400 μ.

The parallel fibres give off as they run along, branches at right angles, which, for the most part, run towards the granular layer; sometimes a branch given off from the upper aspect of the fibre (fig. 16) bends down and runs inwards to form the basket work around the Purkinge cell.

Often one is able to directly trace the passage of a parallel fibre or its branch into the basket work and this structure loosely envelopes the cell and the fibres of which it is formed meet together at some little distance below the invested cell.

As a rule the fibres going to form these baskets widen out and appear flat and tape-like in their passage over the cell (fig. 15).

A number of branches coming from different parallel fibres often meet together in a dense mass as they reach the cell (fig. 17).

In the great majority of instances the fibres of the basket work run over the cell in the same direction as its long diameter, but occasionally transverse strands are seen (fig. 16).

Sometimes instead of a right angle branch of parallel fibre running to form the basket work the fibre itself ends by turning down for this purpose (fig. 17).

The flat tape-like looking fibres often attain a width of 2 or 3 μ, and generally speaking, the whole structure of the basket work is made up of coarse fibres which very loosely envelope the cell. I particularly mention this as Obersteiner (p. 406) speaks of these fibres as " brushes of extremely fine fibres which fix on or closely encircle the aforesaid (Purkinge) cells," although the translator of this work (Dr. Hill) correctly figures the structure in his work on the Golgi method, and it is correctly figured in the latest edition of " Quain's Anatomy."

The basket fibres often envelope the branches of the Purkinge cells some distance from the cell body before passing down to form the basket work proper.

Apart from the above-described system of stout enveloping fibres, there is, both around the Purkinge cell and its main branches, a network of delicate beaded fibrils, very similar in appearance to that seen about the pyramidal cells; whilst, however, it is only occasionally that these delicate fibrils can be detected over the body of the cell, they are comparatively frequently met with over the branches.

Beaded fibrils can also be seen coursing about in different directions in the surrounding matrix, but generally only in the region of the parallel fibres. I have not been able to determine where these beaded fibrils come from; sometimes they seem to be delicate fibres passing off from a basket fibre. Probably, I think, some at least originate from the small cortical cells higher up in the layer, for these generally present a decided but slightly beaded aspect.

W. Aldren Turner and W. Hunter ("On a Form of Nerve Terminations in the Central Nervous System demonstrated by Methylene Blue," BRAIN, vol. xxii., pp. 123-134) failed by the intra-vitam method to detect this delicate lattice work in the cerebellum; and, as by this method, the basket work is also not shown, they argue that this latter is not a nerve arrangement, but neuroglial. I think, however, the fact that it is stained by the present method in such a distinct way and that it can be traced as arising from the darkly stained cells lying in this region will not permit us to accept this view; especially, as the neuroglial elements do not usually stain, and, when they do, they take on a quite distinct colour to that assumed by the nervous elements.

The remaining part of the molecular layer (that is, the outer half), beyond the expansion of the Purkinge cells already alluded to, is occupied by a number of extremely small (9 to 11 μ) very dark cells of different shapes (triangular, square, oval, pear-shaped, &c.), which give off numerous delicate branches on all sides, forming an intricate network in this region. These branches are almost always moniliform or beaded, but the beads are generally only very slight swellings on the fibre, and not nearly so clearly defined as those seen in the cerebrum. The cells are most thickly congregated in the middle third of the layer, but they are met with lying quite close to the surface and also down among the basket fibres near the granular layer, and it is often very difficult or even impossible in this region to distinguish them from the basket cells ; the main points by which we can most certainly differentiate them being the rather larger size and more regular disposition of the basket cells, and the fact that it is these cells only that give off stout smooth fibres running in a parallel direction.

Fig. 10 gives some idea of the relative number and variety in shape of these small cells. Their processes can often be followed for long distances, but their ultimate destination cannot with any certainty be made out, although unquestionably they must be brought into very close relationship with the thorny branches of the Purkinge cells amongst which they lie.

Briefly summarised, the results arrived at from the examination of the cerebellum by this method are as follows :—

(1) It has a selective influence in regard to the staining of the different nerve-cells, some (the Purkinge cells) being very lightly stained, and others (small cells) very deeply stained.

(2) The dark cells can be subdivided into those which certainly envelope the body of the Purkinge cells by their branches and those which, although one is not able with certainty to trace their branches to their destination, there are grounds for supposing invest the branches of the Purkinge cells with a fine beaded network.

In conclusion, I will refer briefly to some inferences that I have been tempted to draw from the foregoing observations.

It seems only reasonable to suppose that the difference in colour, size and shape, &c., of the cells we have met with both in the cerebrum and cerebellum is associated with a difference in their function, and as we have considerable evidence in favour of the pyramidal cells being concerned with efferent impulses. I would suggest that the others—the small dark variety—are concerned with afferent impulses, that in a sense they serve as collectors of ingoing currents which, after passage through them, are distributed to the pale cells with which they are in association.

Although there is considerable difference in the size of the dark cells of the two regions of the brain examined, yet, as they both react similarly to the methylene blue I consider that this, taken along with certain morphological similarities before referred to, will be some justification in associating them together for the purposes of comparison. Now we find that the proportion of dark to pale cells is very different in the cerebrum and cerebellum. In the former they are few in number relatively to the pyramidal cells, in the latter they far outnumber the relatively few Purkinge cells.

Thus we appear to have in the cerebrum a condition the reverse of that found in the cerebellum. In the former there are numerous efferent cells in relation with comparatively few afferent elements, in the latter few efferent in relation with many afferent.

The interpretation that I have given to these facts harmonises with Spencer's theory that the cerebellum is the great organ for co-ordinations in space, while the cerebrum is the organ for co-ordinations in sequence.

DESCRIPTION OF FIGURES.

FIG. 1.

Outline traced from a photograph shows the beaded network over a giant-cell. A beaded fibril is seen passing from (? to) the cell from the upper part of the figure, another from the right-hand side The size and shape of the beads vary; they are generally about 1 μ in diameter, some 2 or even 3 μ. Their distance apart also varies from 1·5 to 8 μ. The nucleus of the cell is shown in the lower part of the figure (× 700).

FIG. 2.

Pyramidal cell with dense network of beaded fibrils encompassing it. Fibrils are seen passing off from the network in all directions. Although the nucleus of the cell is shown, yet in the specimen, when it was in focus, the network over the near surface of the cell was thrown completely out of the field of vision (× 600).

FIG. 3.

Outline, traced from a photograph, shows the network extending along the branches of the pyramidal cell. Coarse fibres can be seen at *a* and *b*, from which delicate fibrils proceed to join the network (× 700).

FIG. 4.

Traced from a photograph, shows a small dark cell, with thin, slightly varicose processes given off from it (× 400).

FIG. 5.

Shows a dark, stout fibre, dividing and giving off from each division fine beaded fibrils to two pyramidal cells. A stout, dark fibre runs up to the left side of the upper pyramidal cell, and apparently also helps to form the investing network (× 600).

FIG. 6.

Also shows a stout fibre dividing and giving off one fine branch to the network about the pale cell shown, while the other passes upwards, probably on its way to another network. This fibril also shows beads on it (× 600).

FIG. 7.

A pale cell encompassed by stout fibres, from which fine beaded fibrils are given off to pass over the cell (× 600).

FIG. 8.

A small, dark, pyriform cell, giving off only a few branches from either end; the lower ones appear varicose and can be followed for a long distance without further branching (× 600).

FIG. 9.

A dark cell with ascending axis cylinder (*a-c*) which gradually gets paler in colour as it passes upwards. Note its smooth contour. From a stout process a fine fibril (*x*) passes off, along which are little irregular swellings, closely resembling the beaded fibrils of a network. The figure is traced from the photograph which was reproduced in my article in the autumn number of BRAIN (× 600).

FIG. 10.

Molecular layer of cerebellum with small cortical and basket cells (× 800).

FIG. 11.

Basket cells and fibres with a Purkinge cell invested by the latter (× 400).

FIG. 12.

Darkly-stained Purkinge cell branch with lateral processes showing thorns. Note the atrophy of the branches (× 600). From a man aged 68 years.

FIG. 13.

Darkly stained Purkinge cell branches with thorns, also a small cortical cell whose branches are shown intermingled with the thorny processes. From a man aged 41 years. Note the stout character of the branches compared with those in the previous figure (× 600).

FIG. 14.

From the same as the last, showing thorns (× 600).

FIG. 15.

Basket fibres spreading out over a Purkinge cell into flat, broad, tape-like strands. Some seem to subdivide longitudinally (× 600).

FIG. 16.

A basket fibre, shown in upper part of the figure, which gives off from its upper side a branch which bends down and goes to help form a basket arrangement around the Purkinge-cell shown in the lower part of the figure. Note the transverse strands and the basket fibres meeting below the Purkinge cell some little distance (× 600).

FIG. 17.

A basket fibre is seen to terminate by bending down to assist in forming the basket work around the Purkinge cell; other fibres are seen coming from other directions and meeting together in a dense cluster to one side of the Purkinge cell, which shows the almost colourless cytoplasm and dark nucleus referred to in the text (× 600).

FIG. 18.

Shows fine beaded fibrils similar to those seen in the cerebrum. Some of these can be seen passing over the Purkinge cell which is just indicated by its slightly darker colouring above the granules (× 600).

A CASE OF FAMILY PERIODIC PARALYSIS WITH A CRITICAL DIGEST OF THE LITERATURE.

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Together with a Report, and Digest of the Literature, on the Pathological Examination of the Excreta.

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THE subject matter of this paper has been divided up as follows:—(1) A description of a case of family periodic paralysis which has been under observation in the wards of the National Hospital, together with a report of the pathological examination of the urine and fæces by Dr. Goodbody, of University College, London. (2) A critical digest of the previous literature of the subject; the part of this relating to the pathological examination of the excreta being written by Dr. Goodbody. (3) A full bibliography. The last two parts have been carried out in some detail, because no case has so far been recorded in this country, and no English paper has hitherto appeared on the subject. The records have as far as possible been carefully examined, with the result that certain cases, such as those of Cavaré, Romberg, Gibney, Bernhardt, and Burr, which have previously been included as examples of this disease, but which appear to differ from it in essential features, have been excluded from the bibliography. References to these cases may be found in an excellent paper by Dr. E. W. Taylor in the *Journal of Nervous and Mental Diseases*, New York, for September and October, 1898, to which I am indebted for all the earlier references.

I take this opportunity of expressing my thanks to Dr. Howard H. Tooth, under whose care the patient was admitted to the hospital, for permission to publish this case, and also to other members of the honorary medical staff for kind assistance and advice.

My thanks are also due to Dr. J. K. Mitchell, of Philadelphia, and Dr. J. J. Putnam, of Boston, for sending me reprints of their papers on the subject, and to the former for kind suggestions as to lines of investigation and treatment.

T. L., aged 16, was admitted into the National Hospital on October 12, 1900, complaining of attacks of loss of power for about two years.

Family and personal history.—There is absolutely no history of any similar trouble nor of any neuropathic tendency in his family so far as can be discovered. Patient's mother died shortly after the onset of the lad's attacks from morbus cordis, the result of acute rheumatism; his father is a temperate man, but is said also to have heart disease, though he has not had rheumatism. Two sisters are living, strong and healthy; a third died of scarlet fever and diphtheria. Patient was the third child in a family of four, and was quite healthy as an infant, but somewhat backward in walking, not doing so properly until 5 years of age. He is said to have had smallpox as a child, but this seems doubtful, as he bears no scars, and has four good vaccination marks, which were made when he was a baby. At the age of 8 years he had a sudden attack of "cramps" in the legs when out walking with his mother, but nothing further is known of this incident. Whilst at school he tried to learn to swim, and liked the water, but had to give it up because he invariably got cramp in the legs. Apart from the above incidents he has always been very healthy. His habits tend rather to an out-door life, and he is very fond of exercise, but he did fairly well at school, passing the sixth standard at the age of 13. He has always been of a very excitable temperament, and is at times inclined to be violent; his sister asserts that an attack of paralysis always follows one of these outbursts of wild excitement, although the latter are by no means frequent precursors of the attacks. He worked for twelve months after leaving school at a lead-pencil factory, but since the onset of his present illness has had to give up regular work, and has acted as an errand boy. He has lived in fairly comfortable circumstances all his life in Battersea.

History of illness.—The first attack of paralysis occurred about two years ago at the age of 14, the second four to six weeks later, and since then attacks have followed at somewhat irregular and gradually decreasing intervals until now they occur about once a week, although a few weeks ago he had as many as three in five days. The onset has always taken place at night, without any known cause or warning, and he is absolutely certain that he is not more liable to an attack after a severe day's exercise than at other times. He accepts the attacks philosophically, his only trouble being that they prevent him from getting permanent employment. The attacks are all very much alike in character, differing only in severity and duration; he never loses consciousness, has never been unable to talk, and suffers only from the discomfort of lying for a long time in one position. On three separate occasions, however, he has had alarming difficulty in breathing, and on the last of these he also had great difficulty in swallowing. During an attack he does not feel any desire for food, but will sometimes drink freely or eat fruit; he is unable to pass urine, but does not feel any desire to do so, and his bowels are never opened until after the attack, even if it lasts for two days. During the first twelve months of his illness he vomited several times soon after the onset of an attack, but this has not occurred lately, though he often feels some nausea.

On admission to the hospital on October 12, 1900, he presented a rather feeble appearance, with very little subcutaneous fat and muscles somewhat poorly developed. Mentally he is bright, cheerful and intelligent, and a fair witness. On physical examination, the only abnormalities noted were that the right pupil was a little larger than the left, both reacting normally, and that on lateral deviation of the eyes, there was some irregular nystagmoid jerking, more marked towards the right than the left. Muscular power was everywhere proportional to development, and sensation was normal; all deep reflexes were present, normal and equal on the two sides; abdominal, cremasteric, and plantar skin reflexes also normal, the last of flexor type.

The electrical reactions of muscles were examined on several occasions in the intervals between the attacks, and found to be absolutely normal in every respect.

Description of the Attacks.

Many have been observed whilst in the hospital, but they are all so much alike that it is unnecessary to describe more than one; the differences merely depend upon the severity and duration of the paralysis. This attack is chosen because it was observed practically from the onset, which took place in the afternoon, patient having been kept in bed for purposes of observation. He had been feeling perfectly well, and, as usual, had no warning whatsoever; he was lying talking to some friends about 3 p.m. when he noticed a "dull aching" about his knees, and found, on moving them, that his legs were weak; the aching was at once relieved by change of position. He also found, on trying to sit up, that he had great difficulty in doing so. His hands and arms were at this time quite strong.

At 4 p.m. The weakness in the legs had very considerably increased, but he could still draw them up in bed, while extension of hips and knees was distinctly better; he could not sit up in bed without assistance. The arms were distinctly weak in the proximal joints, but the change was not nearly so marked as in the legs.

At 4.30 p.m. The legs much in the same condition, but grasp as tested with dynamometer was distinctly less.

At 5.30 p.m. *Upper extremities.* All movements present, with the exception of flexion of the elbow on the right side, but all are feeble. The best movements are those of the fingers and wrists, the left a little stronger than the right. *Lower extremities.* Flexion of hip and knee and adduction of the thighs are now impossible; all other movements are still present, but are very feeble, the best of all being those of the toes. *Neck* muscles are also weak, although the head can be still raised off the pillow, but not against the slightest resistance, while extension of the neck is more powerfully performed. *Trunk* muscles can all be voluntarily contracted, but all are feeble.

The muscles supplied by cranial nerves are quite normal.

Electrical reactions.—No reaction to either faradism or galvanism could be obtained in the hamstrings or abductors of the thighs, nor in the right biceps humeri. The quadriceps femoris reacts to both currents, but less readily than normal; the contraction shows no qualitative or polar change. Currents of varying degree, proportional to the amount of voluntary power are required in other muscles. Prolonged tetanisation of the flexors of the fingers of the left hand with a faradic current was maintained, but no diminution of reaction was observed, such as is seen in myasthenia gravis, nor did the subsequent increase in paralysis in the muscles so treated show any difference from that of the opposite side.

Mentally he is perfectly natural, talks normally, and complains of nothing except the discomfort of being unable to change his position.

No change in objective sensibility to any form of stimulus could be detected.

At 7 p.m. he managed to eat a little food, but was unable to feed himself.

At 7.30 p.m. He is quite unable to move the head or shoulders; on sitting him up he simply doubles up and his head falls either forward or backward without his being able to control it in the least. Respiration is almost entirely diaphragmatic; coughing is almost impossible as the abdominal muscles only just contract in the attempt and only the very feeblest movement is perceptible in the latissimus dorsi on either side. All arm movements are very small, flexion of the elbow and extension of the wrist being quite absent on the right, but just present on the left side where extension of elbow and flexion of wrist are quite absent. The fingers on both sides can still be flexed, extended, adducted or abducted though with small power, and the grasp is practically nil. In the lower extremities slight extension and ext. rotation of hips is still possible and the quadriceps can just support the weight of the lower leg. Feeble movements of ankles and toes are present.

Reflexes.—Knee-jerks are present but only a very feeble contraction is obtained, though it is noticeable that this is very ready and is not reinforceable. All other deep reflexes are unobtainable. Abdominal and cremasteric skin reflexes absent. Plantars still ready but the movement of the toes is small and flexor in type.

The muscles supplied by the cranial nerves are still normal.

No change mentally or in sensation. Pulse good volume and tension, quite regular. Patient had not passed urine since morning, but there was no obvious distension of the bladder and he had no desire to micturate, but after severe effort he succeeded in passing nine ounces, and about one minute later, after further straining, he passed one ounce more.

9.20 p.m. Sleeping quietly till now. He is bathed in perspiration from head to foot (this was not so ten minutes ago). The face and skin generally is flushed and feels hot to the hand. Temperature 98° in the mouth. Pulse 96 regular, tension low. Respiration 18 regular and easy but purely diaphragmatic. He cannot take a deep breath and cannot cough. In the upper extremities the only movement now possible is a very slight one in the index finger on either side. The right leg is completely paralysed, but in the left a slight contraction can be made in the quadriceps not sufficient to move the leg, while a very faint contraction in the vastus internus can be produced by tapping the patellar tendon. On the left side also he can just move the big toe and here also a faint plantar reflex (flexor) can be obtained. No other reflexes obtained. Cranial nerve supply normal. Sensation normal.

10 p.m. Sweating has ceased, but the skin is still flushed. Left leg now quite paralysed with the exception of slight movement of big toe with faint plantar reflex still present, knee-jerk quite absent.

11.30 p.m. Some improvement in arms; can now flex fingers and wrists slightly and also pronate and supinate faintly; there is also a very feeble contraction in left triceps humeri with faint triceps-jerk. Right leg completely paralysed with absent reflexes; slight power in left quadriceps femoris and big toe with very faint knee-jerk and plantar reflex.

Coughing and deep breathing are impossible.

The paralysed muscles, although the palsy is of the flaccid type, do not show any degree of hypotonicity and feel firm and solid to the touch; none of them contract to direct percussion.

He now shows for the first time some partial bilateral ptosis, the upper lids cannot be raised above the upper edge of the pupil. The right pupil is markedly larger than the left and has certainly dilated; both however react normally to light and in accommodation. Ocular movements are good in all directions, but still show the nystagmoid jerkings noticed on admission. Facial muscles, masseters, palate and tongue move normally. Swallowing and speech normal.

12.45 a.m. Arms are steadily improving and the biceps and triceps on both sides contract feebly, while their tendon-jerks have returned. Shoulder girdle muscles and those of extraordinary inspiration are still paralysed. Slight movement of toes on right side now present, with return of right plantar reflex (flexor), otherwise legs much as before. The ptosis has very nearly disappeared.

2 a.m. Slight further improvement. Biceps, triceps and supinator-jerks present on both sides. Right knee-jerk absent; left faint. Achilles-jerks: right faint, left absent. Ptosis quite gone. Patient now sweating profusely and skin much flushed and feels hot to the hand although the temperature in the mouth is only 97° F.

3.15 a.m. Some power now in all muscles, best in periphery of all limbs. All reflexes are present, but still feeble.

4.30 a.m. Much improved; he can now move all limbs and head, but not yet able to sit up. Feels well but rather stiff and sore, as if he had done some hard exercise.

10.30 a.m. Power now everywhere good, though still some feeling of stiffness.

The above represents a moderately severe attack, and may be taken as a type. All the severe attacks have come on while the patient has been in bed, and vary in duration from five to twenty-eight hours, generally lasting about twelve to sixteen hours. He however describes one attack that lasted more than two days and nights. During the daytime he has on several occasions complained of stiffness and weakness of the knees and sometimes also of the hands, and on examination his gait is distinctly paretic and his arms weaker than normal, but these have invariably passed off in the course of an hour or so, whether he has taken exercise or has rested. On two occasions while in hospital, when recovering from an attack, which had lasted seven or eight hours, he has relapsed before recovery was complete, into a more severe degree of paralysis than before, the relapse running the usual course and lasting about twelve hours.

Order of involvement of muscles during an attack.—This is not by any means constant as regards individual muscles, but certain rough generalisations can be made. The weakness has always appeared first in the lower extremities, and very soon affects lower trunk muscles; it next spreads to upper arm and trunk, involving the neck about the same time. As a rough rule it may be stated that the more proximal limb muscles are the first to be affected, the muscles of the digits being always the last to become completely paralysed. The muscles which have, in most attacks, been the earliest to lose power completely are the hamstring and adductors of the thighs, the quadriceps being affected much later. But this is not constant; in one attack the arms became completely paralysed, including even the fingers, when he was still able to move the legs, although on this occasion, too, the weakness had begun in the legs quite as early if not earlier than in the arms. It has not been possible from the order of spread to attribute the affection to a progressive involvement of spinal anterior cornual cells. The only cranial nerve muscles that have been affected whilst in hospital are the levatores palpebræ and the right iris oculi. These have been affected on each occasion only during the height of the paralysis.

The intercostals and extraordinary muscles of inspiration are affected early and become completely paralysed; the diaphragm, however, continues to act, although I am inclined to think that it has been to some extent weakened in some of the most severe attacks, because the power of taking a deep breath, and even of altering his shallow breathing in depth at all is entirely lost.

The order of recovery is for all practical purposes exactly the reverse of that in which they were affected, that is to say the last muscles to lose power are the first to regain it.

On several occasions marked changes have been observed in the condition of the *heart.* The area of superficial cardiac dulness becomes markedly increased, and diffuse pulsation over the præcordial area, including the second and third spaces to the left of the sternum. The first sound becomes soft and not clear, but I have never heard a definite murmur either at the apex or base; the second sound at the left base is accentuated and reduplicated, while the first aortic sound is feeble. The pulse becomes soft but fairly full and distinctly intermittent. On one occasion breathing was seriously affected and mucous rattling was audible in the larger tubes.

Repeated *electrical examinations* reveal the following facts :—In the intervals between the attacks the muscles all react normally. During an attack there is a gradual reduction of excitability both to faradism and galvanism *pari passu* with the loss of voluntary power, until with complete palsy there is complete loss of excitability to both currents. With the reduction there has never been any change either in quality of contraction, nor has there been any polar change to galvanic stimulation. As voluntary power recovers there is a gradual return of excitability to both currents.

The facial muscles have always reacted normally when stimulated through the nerve or directly even at the height of an attack.

Excitability of muscles to percussion which, during the intervals is normal, disappears at the height of paralysis.

The *reflexes* disappear *pari passu* with the loss of power It, is, however, noteworthy that both with tendon-jerks and skin reflexes, although the amount of contraction produced by the stimulus diminishes with the loss of voluntary power, yet the reflex is still as readily obtained as before, and with only the same stimulus, so that so long as there is the least power of contraction in the muscles a ready even if slight reflex can be obtained ; moreover it can be elicited with the smallest return of voluntary power. Another point of interest is that the contractions cannot be increased by reinforcement.

The *temperature* during an attack has been several times taken at short intervals and has been found to fall slowly to 96° or 97° F., but exactly the same fall occurs quite regularly when taken during a night in which the patient is not having an attack. If the temperature is watched during an attack coming on in the day time no fall in temperature is observed. No rise has been noticed at any time.

Relation of Attacks to Muscular Exercise.

Much stress has been laid by some authors upon the excitation of attacks by severe muscular exercise, but both from the history as given by the patient and from observations while in hospital, this has certainly not been the case in T. L. When first admitted he was kept completely at rest in bed for six days, during which time he had two severe attacks, lasting fourteen and sixteen hours respectively, and also two slighter ones, each lasting five or six hours. He then objected so strongly to being kept in bed that he was allowed to get up and take a moderate amount of exercise, working about the ward and occasionally going for a walk. During the next six days he had only two attacks—one severe, the other slight. In the next ten days he had, however, six attacks, two of them severe and four moderately so. At the end of that time he used a Whiteley's exerciser twice a day, as well as doing the same work as before. The attacks then diminished to about one per week on an average, sometimes more, sometimes less. At a later period, when he had been having attacks about once a week, he was again kept in bed for a week, and during that time he had one severe and two slighter attacks.

It has also been stated that attacks could be postponed after the appearance of the first signs of weakness by taking exercise, but that the subsequent attack which follows as soon as the patient goes back to bed is more severe in degree. It has not been possible to test this in regard to T. L., as the rule is for him to wake up at night so far paralysed that exercise is practically out of the question. He, however, gives a history of having, on waking in the morning and finding himself weak, succeeded in "walking it off"; but it is clearly open to question whether he is not in reality just recovering from, rather than just beginning, an attack, and he is very certain that this "walking it off" has not in any case been followed by a more severe attack.

Relation of Attacks to Gastro-intestinal Functions.

Some of the recorded cases have shown a relation between the action of the bowels and the onset of an attack, but no such relation has been observed in this patient. His bowels are usually opened once or twice a day, and this has been so during the whole of his stay in hospital; he has never suffered either from diarrhœa or constipation.

An attempt was made to determine whether alterations in the diet produced any effect on the frequency and severity of the attacks; and for this purpose he was kept for a time on a carbohydrate-free diet, and subsequently given large doses of glucose, together with the ordinary full diet, but neither alteration seems to have had any effect. On the other hand, there are certain indications of some disturbance of gastro-intestinal function in his history of vomiting and nausea in the early stages of an attack at the beginning of his illness, and whilst in hospital he has on several occasions complained of nausea, but has not vomited. The tongue, also, in the attack is generally a little furred. The examination of the stools and urine has been carried out by Dr. Goodbody, and his report and conclusions are appended below.

Relation of Attacks to Urinary Excretion.

Acting upon the hypothesis, which is more fully discussed below under the heading of the pathology of the condition, that the exciting cause of an attack may be the failure to completely remove the toxic products of metabolism from the body, an attempt was made to ensure more thorough elimination by the production of diuresis. The method employed was the administration of large doses of soda-water and Imperial drink in addition to the ordinary amount of fluid taken with meals, and at the same time small doses of digitalis and potassium acetate. Previous to this treatment he had been having attacks at the rate of at least one per week; in the following three weeks he had no attack at all. In order to control this result, the amount of fluid ingested was then diminished to two pints all told, and the digitalis and potassium acetate omitted; but he had only two abortive attacks during the next fortnight, although the amount of urine voided averaged about twenty ounces less than before. He was then kept absolutely at rest in bed with the same diminution in the fluid part of his diet; on the following day he had an abortive attack, on the next a more severe one, and on the third day a still more severe attack with dilatation of the heart. The amount of fluid taken was then increased as before, and the mixture of digitalis and potassium acetate again administered, with the patient still at complete rest in bed; in the following six days he had no attack at all, even of an abortive character.

These observations are very striking, especially when it is remembered that on the two previous occasions on which he was kept in bed for six days he had four and three attacks respectively, and should they be confirmed by other observers, ought very materially to help us to a true conception of the nature of this extraordinary disease.

The Blood.

The blood has been examined, both in the free intervals and also during the height of an attack, but the number of red and white cells, amount of hæmoglobin, and the proportion of the different varieties of white cells has been found to be in each case well within the limits of the normal. No abnormal varieties of red cells, and no foreign elements have been seen in any of the blood films microscopically.

Muscle.

A portion of the L. Sartorius was excised, when that muscle had almost completely lost its power of voluntary contraction in an attack, from which sections and photographs were kindly prepared for me by Dr. J. S. Collier, pathologist to the hospital. The great majority of the sections showed the condition represented in fig. 1, in which is seen the most extraordinary amount of fissuring in the individual muscle fibres, such as is certainly not seen in sections of normal muscle, prepared in the same way, viz., by hardening in Müller's fluid and embedding in paraffin. Sections hardened in formalin showed precisely similar changes. Fig. 2, however, represents photographs of sections of the muscle hardened in Müller's fluid, and embedded in celloidin. In these, although not entirely absent, the fissuring is comparatively very slight, and this fact tends to show that the fissuring is probably artefact in nature. In spite of this, one cannot but believe that there must be some essential abnormality in the muscle fibres to produce such uniform and unusual appearances.

Pathological Examination of the Urine and Fæces
(by Dr. Goodbody).

During one of the attacks, it was decided to investigate the condition of the urine in order to see how far it agreed with what was described by Crafts. For this purpose, the

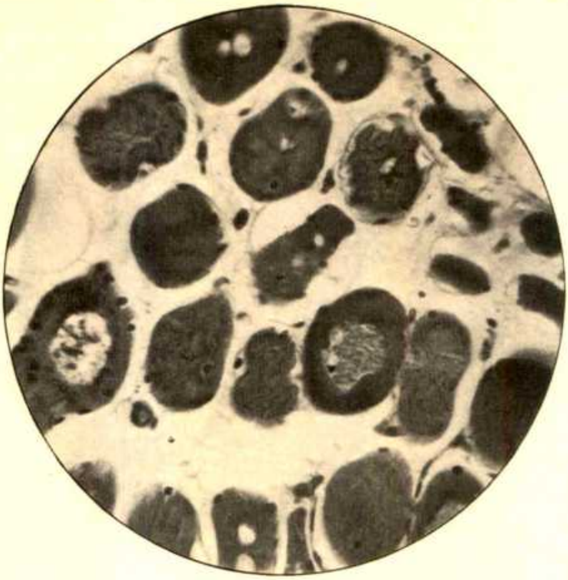

FIG. 1.

Transverse section of left Sartorius muscle excised during an attack, hardened in Müller's fluid and embedded in paraffin. In the centre of many of the fibres is seen a more or less circular fissure some of which contain some hyaline material.

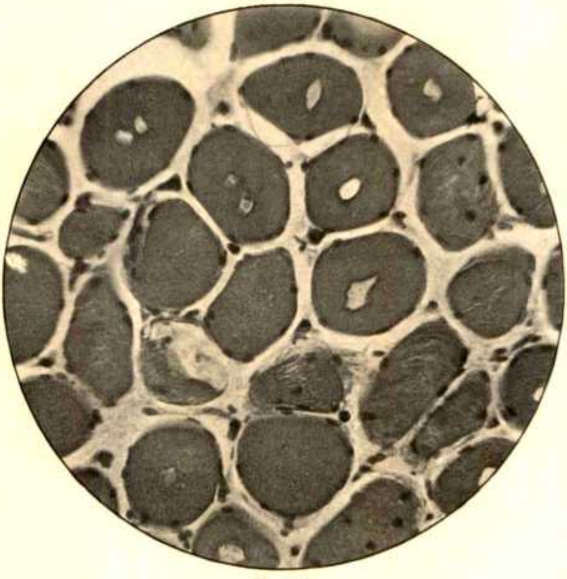

FIG. 2.

Transverse section of a portion of the same muscle hardened in Müller's fluid and embedded in celloidin. It will be seen that the fissuring although still present is much less marked.

total urine passed during the twenty-four hours in which the attack occurred was collected, and also that excreted during the succeeding twenty-four hours. For the purpose of comparison, a two-day period was taken about a fortnight later, in the interval between two attacks, and the urine of each day submitted to analysis in order to obtain a result as nearly approaching the normal as was practicable.

		Period of Attack.			Interval.		
URINE.	Quantity	430	1230	830	660	1660	1160
	Specific gravity	1020	1026	1023	1028	1019	1021
	Nitrogen	6·62	18 01	12·32	7·42	18·43	12·93
	Urea	11·91	31·88	21·90	14·52	32·44	23·48
	Uric Acid	0·31	0·90	0·61	0·32	0·85	0·58
	Chlorides	—	—	—	—	—	—
	Phosphates	—	—	—	—	—	—
Sulphates.	Total	0·77	4·73	2·75	1·60	3·92	2·76
	Alkaline (A)	0·69	4·40	2·55	1·48	3·69	2 59
	Aromatic (B)	0·08	0·33	0·20	0·12	0·23	0·17
	Ratio A : B	8·6.1	13 3:1	12 7:1	12·3:1	16 0:1	15·2:1

In dealing with the first period, that of the attack, it must be pointed out that on the first day the patient practically took no food, as the seizure lasted almost twenty-four hours. On the second day, he took a considerable quantity of milk. The quantity of urine excreted during the first day was small, amounting only to 430 cc. with a specific gravity of 1020. On the following day, the excretion rose considerably, as 1230 cc. was passed, while the specific gravity, notwithstanding the increase in the quantity of water eliminated, rose to 1026.

On the first day, the quantity of total nitrogen excreted in the urine was very low, only 6·62 grammes, while on the second day, 18·01 grammes were eliminated. Although, as already mentioned, some milk was taken on this day, still the quantity excreted was very large in comparison with the preceding twenty-four hours, and there appears to have been an increase in the metabolic processes, especially as 82·61 per cent. of this amount was excreted in the form of urea. On the first day also the proportion of the total nitrogen excreted in the form of urea was about the normal, being 83·96 per cent. The quantity of urea excreted on each day was 11·91 grammes and 31·88 grammes respectively.

The amount of uric acid excreted on the first day was 0·31 grammes, being about the normal quantity for a boy of his age. On the second day, there was a considerable increase in the quantity found (0·90 grammes), although it will be noticed that the amount of total nitrogen excreted in this form remained about the same.

On turning to the consideration of the sulphates in the urine, the small amount of proteid metabolism occurring in the organism on the first day is shown by the fact that only 0·77 grammes of total sulphates were eliminated. Of this amount, 0·69 grammes were excreted as alkaline sulphates, and only 0·08 grammes in the aromatic group. The ratio of the former to the latter was 8·6 : 1. On the second day, the great activity of the proteid metabolism is shown by the large quantity (4·73 grammes) of total sulphates excreted in the urine. 4·40 grammes of this amount was eliminated in the form of alkaline sulphates, while the quantity eliminated as aromatic sulphates was rather above the normal, and was 0·33 grammes. The ratio of the alkaline to the aromatic sulphates was 13·3 : 1.

On the two days of the second period, there was a great similarity in the excretion of the urine, 660 cc. being passed on the first day, and 1,660 cc. on the second day. The specific gravity, however, reverted to the normal course, diminishing from 1,025 on the first day to 1,019 on the second day.

The excretion of total nitrogen was also low on the first day, and only amounted to 7·42 grammes, and the proportion eliminated in the form of urea was high, being 91·32 per cent. On the following day the same marked increase in the quantity of total nitrogen excreted was observed, as 18·43 grammes were eliminated, the amount passed in the form of urea being 82·13 per cent. It should be mentioned that the patient took his food well throughout this period. The quantities of urea excreted were 14·52 grammes and 32·44 grammes respectively. The same diurnal differences were also observed in the elimination of uric acid, 0·32 grammes being eliminated on the first, and 0·85 grammes on the second day.

The lessened activity of proteid metabolism is well shown by the small quantity of total sulphates found on the first day in the urine (1·60 grammes). 1·48 grammes of this amount was eliminated in the form of alkaline sulphates, and only 0·12 grammes in the aromatic group. The ratio of the former to the latter was 12·3 : 1. The quantity of total sulphates excreted on the second day was 3·92 grammes, so that the metabolic activity of the organism was much increased. Of this quantity 3·69 grammes were eliminated as alkaline sulphates, and 0·23 grammes in the aromatic group. The ratio of the alkaline to the aromatic sulphates was 16 : 1.

From the above, it is seen that there was a great similarity between the two periods, although the quantities found were slightly larger in the second period.

If the averages of the two periods be compared, it will be seen that no indications as to either diagnosis of the attacks or to treatment can be drawn, since the various substances analysed for give practically the same results. The principal difference is that the average excretion of urine per diem was 830 cc. in the first period as compared with 1,160 cc. in the second period. This appears to give a clue to the appropriate treatment, namely, diuresis, especially if—as might be concluded from the analysis of the first day of the second period—the patient was on the verge of an attack. The average specific gravities were rather above the normal, being 1,023 and 1,021 respectively.

The average excretion of total nitrogen was practically the same, 12·32 grammes and 12·93 grammes, while the proportion eliminated in the form of urea was 83·29 per cent. in the first period, and 86·73 per cent. in the second period. Despite the daily variations observed in the excretion of uric acid, the averages of the two periods (0·61 grammes and 0·58 grammes) agree closely.

On turning to the consideration of the total sulphates in the urine, it will be seen that the average of the first period was 2·75 grammes, and of the second period, 2·76 grammes. Of the above quantities, 2·55 grammes were eliminated as alkaline sulphates in the first period, and 2·59 grammes in the second period, so that these averages also closely agree.

The aromatic sulphates averaged 0·20 grammes and 0·17 grammes respectively, and the average ratio of the alkaline to the aromatic sulphates was 12·7 : 1 in the first period and 15·2 : 1 in the second period.

Neither albumen nor sugar was found in the urine on any of the four days analysed.

Opportunity was also taken on each day to test the toxicity of the urine according to Bouchard's method. The urine of the day on which the attack occurred was injected into the external jugular vein of a rabbit weighing 1·750 kilos., which had been previously anæsthetised, and death occurred after 58 cc. had been injected, so that 31·1 cc. were used per kilo. body weight. In this case, the secondary dilatation of the pupil usually observed was very slight, and there was also no diuresis. Previous to death, only extremely slight convulsions were observed, and coma lasted to the end, as the heart apparently was paralysed before the convulsions reached their ordinary severity. The small number of cc. used per kilo. indicates that some toxin of a paralytic nature was present in excess.

110 cc. of the urine of the following day was injected into the same vein of an anæsthetised rabbit, weighing 2·250 kilos., the number of cc. per kilo. body weight being 49. In this case the customary sequence of events was observed, namely, contraction, followed by extreme dilatation, of the pupil, diuresis, and, immediately before death, several well-marked convulsions. There is, therefore, evidence of increased toxicity of the urine of this day also, though not so marked as on the previous day.

On the first day of the second period, a rabbit weighing 1·850 kilos. was used, and canula was inserted into the external jugular vein, after the animal had been anæsthetised. On this occasion, 121 cc. were used, and therefore, 65 cc. per kilo. body weight was necessary to cause death. That is to say, about the normal quantity, according to Bouchard's statement. The customary phenomena were also observed in this case.

Owing to an accident, we were unfortunately unable to make a similar test with the urine of the second day of this period.

From the above results, it will be seen that in this patient there was a great increase in the toxicity of the urine during the attacks, and that the toxin or toxins were of a paralytic nature; but no attempt was made to isolate it or them.

Following the procedure given by Crafts (18), extracts of the fæces passed immediately after an attack and in the interval were made by Brieger's and Otto's methods and the results obtained were as follows:—

One gramme of each extract of the fæces passed immediately after an attack was injected into the subcutaneous tissue of a guinea-pig and the animal was carefully observed for the ensuing forty-eight hours. No paralytic symptoms whatever were observed, and all the animals appeared perfectly well with the exception of one which died from sepsis one week later.

Of the animals, injected similarly with extracts of fæces passed during a healthy interval, one died almost immediately, but none of the others showed any symptoms at all.

CRITICAL DIGEST.

Historical.

The first undoubted case of this disease was published by Hartwig (1) in 1874, under the title of "Intermittent Spinal Paralysis," in his inaugural address at Halle. He gives no details as to family history, but notes that during the attacks the electrical excitability of the muscles is almost entirely suppressed. A probable case was published without much detail by Samuelsohn (2) in 1876. In 1882 the first evidence of the familial nature of the disease was noted by Schachnowitsch (3), who published a case whose father had been similarly affected. In 1885 appeared a most excellent monograph by Westphal (4), giving a detailed account of the clinical phenomena to which but little has been added since. In this paper we also find the first mention of temporary dilatation of the heart. The following year Cousot (5) published the first instance of marked family affection, and gave a digest of previous papers. This was rewritten and amplified in 1887 (6).

The next paper of importance was that of Goldflam (8) in 1890, which is preliminary to a more detailed account of the same material which appeared in 1891 (10). This is very full and deals largely with the electrical phenomena, and also includes a very excellent analysis of all previously recorded cases. Oppenheim (11) in 1891 gives another full account, particularly of the electrical reactions of the case published by Westphal in 1885. Goldflam (13, 14) contributes two further papers in 1895 and 1897, giving some fresh cases, and also the results of investigations carried out with a view to determining the ætiology of the condition.

In 1898, Taylor (15) published two cases belonging to a family in which the disease dates back for five generations, and gives an excellent *resumé* of the whole of the previous literature, and it is to this paper that I am indebted for all the earlier references. Since that time three papers by American authors have appeared, each publishing new cases. Mitchell (16), 1899, carried out some important examinations of the blood, urine and fæces, but with negative results. Putnam (17), 1900, gives most of his article to the discussion of the question of inhibition; and lastly, Crafts (18), 1900, asserts that he has found a toxin in the fæces, which on injection into animals produced temporary paralysis; but he does not appear to have controlled his experiments.

Besides the above papers, cases have been recorded by Greidenberg (7), Pulawski (9), and Hirsch (12).

Clinical Phenomena.

The clinical manifestations of this disease are so constant and similar in all cases that it is unnecessary to describe them again as a whole; the case described above will serve well as a type. But the main features may be briefly summed up as follows: Periodic attacks of flaccid paralysis in an otherwise healthy individual, of varying duration from five or six to seventy-two hours, during which there is a gradual and progressive onset, leading in complete attacks to absolute paralysis of all four extremities, neck and trunk, but not affecting, as a rule, the muscles supplied by the cranial nerves. Proceeding *pari passu* with this loss of power there is loss of muscular excitability to electricity and percussion, and abolition of all reflexes in the affected parts. After a variable period this condition gradually disappears, and the affected person once more becomes apparently sound and healthy. There is never at any time any loss of consciousness or impairment of mental functions, nor any demonstrable change in any afferent path.

Some of the important points may now be discussed in more detail.

Age of onset of first attack.—This has varied between 6 and 24 years; in one case, recorded by Goldflam, the mother of two children similarly affected, had only one attack, and this occurred at the age of 36 years, but if we exclude this the average age at which the disease has appeared, in the cases where this point is definitely stated, is 14 years.

Sex.—The two sexes are about equally affected.

Time of onset.—This has been almost invariably at night, the patient waking after a variable period of sleep to find himself more or less paralysed. Attacks, however, do sometimes start in the daytime, especially, as I have found, if the patient is kept continuously in bed. Mitchell obtained the history that one of the ancestors of his patients had sometimes to get off his horse when riding, and "have a paralysis." Besides these attacks, what appear to be abortive attacks have been noted by several observers as occurring during the daytime, consisting merely of slight weakness of the legs and possibly arms, lasting half to one hour and then passing off altogether. Possibly some of the attacks reported as having been walked off were of this nature.

Periodicity.—The intervals between the attacks as a rule are irregular; at first amounting to months, they may steadily diminish until the attacks occur every week or even oftener, and this condition obtains well on into middle life, when the intervals tend again to increase.

Premonitory symptoms have been in the majority of cases entirely absent, but Westphal and Cousot describe pricking in the legs, formication, muscle pain and thirst as occurring before the onset of an attack. In one of Goldflam's cases there was itching on those evenings when no attack occurred, coming on about the usual time for the onset of the attack, but on the evenings when he had an attack no such itching was felt. Hirsch describes a heavy tired feeling in the legs as occurring for some days before the onset of an attack. In the later recorded cases no such prodromata have been observed.

Order in which the muscles are involved.—This appears to be practically the same in all cases, the weakness beginning in the legs, extending then to the arms, and lastly to the trunk and neck. The more distal muscles, such as those of the fingers and toes are always the last to become completely paralysed. Cousot records one case in which the attacks began in the arms on a few occasions, and in one of Goldflam's cases, too, the order was hands, arms, legs. The arms were most, though not first, affected in one attack in my own patient. The order of recovery appears to be the reverse of the order of onset.

Cranial nerves have been unaffected in nearly all cases, but in some of the very severe attacks changes have been observed. One of Taylor's patients gives a history of a somewhat doubtful attack which was preceded by loss of consciousness, and said to be due to taking "some headache medicine." In this attack, which was very severe and lasted three days, the face was involved. Crafts records difficulty in opening the mouth, Hartwig and Cousot difficulty in speaking and swallowing. Hartwig also observed inequality of the pupils and even myosis. In my own case partial bilateral ptosis and dilation of the right pupil have occurred twice while under observation.

Respiration is always affected in complete attacks, in that the intercostals and extraordinary muscles of inspiration are paralysed. The diaphragm, however, seems to escape, but it seems probable that it is weaker than normal, because, as noticed by most observers, respiration is very shallow, and there is complete inability to deepen the breathing. Goldflam records that on one occasion there was dangerous asphyxia, and in the doubtful attack alluded to above in Taylor's patient, artificial respiration had to be performed during one night.

Heart.—The affection of this organ has been now recorded by so many observers that there can be no doubt as to its occurrence. This point is one of very great importance as giving some indication of the seat of the change which results in the paralysis. The first allusion to any change in the condition of the heart was made by Westphal, who described slight temporary enlargement. Oppenheim had the opportunity of examining this patient again six years later, and described the heart in some severe attacks as being irregular in action, with a systolic murmur at the apex and slight accentuation of the second sound at the base, while the first sound was feeble and the pulse slow; there was, however, no enlargement of the area of cardiac dulness. Since then Goldflam and Mitchell have found systolic murmurs without enlargement of the area of cardiac dulness. Hirsch and myself have found enlargement of the area of dulness, with some impurity of the first sound at the apex, but no definite murmur. Crafts describes the pulse as being sometimes dicrotic, but with no murmur or cardiac enlargement. From these observations it will be seen that the change consists in dilatation with or without definite regurgitation through the mitral valve.

Electrical changes.—The loss of excitability of muscles to both faradism and galvanism which takes place during an attack has been confirmed by all observers, and is one of the most characteristic features of the disease. Mitchell added to our knowledge of the electrical condition by using needles thrust directly into the muscles as electrodes, and found that even then a very powerful current is needed to produce any contraction. This point will need further discussion later under the pathology of the disease. All observers agree that the change is purely a qualitative one and that it is proportional to the loss of voluntary power.

In the intervals between the attacks the majority of patients have reacted quite normally to both currents directly and indirectly. Goldflam, however, has found permanent changes. In two brothers affected with family periodic paralysis he found partial R.D. in the small muscles of the hand, *i.e.*, diminished faradic excitability, increased galvanic excitability with A.C.C., equal to, or even greater than K.C.C. He lays great stress upon this as showing some definite changes to be constantly present in the muscles; but the fact that these particular muscles were not specially affected during the attacks, either with respect to early onset or degree of paralysis attained, hardly bears out this supposition. The vast majority of evidence points to the electrical change, which takes place in the attacks, being as temporary as the loss of motor power; both being probably due to the same temporary cause.

Ætiology.

Heredity.—The familial nature of the disease is forced upon one's notice by the cases recorded by Cousot, Goldflam, Taylor and Mitchell, who give the numbers of relatives affected as five, nineteen, eleven and seven respectively—a total of forty-two cases in four families. Goldflam and Taylor have both traced the disease back through five generations. Of the remaining cases three members in one family have been recorded by Goldflam and two by Schachnowitsch and Hirsch, while all others appear to be sporadic. Transmission of the disease takes place through both males and females, and sometimes spares one generation to reappear in the next.

Heredity thus plays a most important part in the causation of the disease, but it has not been shown that the affected families are subject to any other form of hereditary neurosis or myopathy.

Specific fevers have been suggested as exciting causes of the disease, especially among the earlier writers. Malaria more particularly was quoted, probably because of the periodicity. The majority of patients, however, have never suffered from malaria. Scarlet fever and typhoid have been noted in the previous history of some cases, but although they might possibly act as the exciting cause of symptoms of this disease, there is no evidence to show that they have any effect in producing the disease itself.

Exciting causes of attacks.—In all cases attacks may come on without any assignable cause, but three conditions have been quoted which have appeared to precipitate attacks.

The first and most important of these is physical overexertion, and it has been so strongly supported by nearly all observers, that there can be little doubt as to its accuracy. In Mitchell's case, and in the one described above, no such connection could be demonstrated.

This question will be further discussed under the pathology.

Indiscretions in diet and gastro-intestinal trouble have also been accredited with the power of exciting attacks. Hirsch's patient attributed his to the drinking of tea or spirits overnight, while Crafts states that his patient had learnt to eschew jams and cheese. Careful observations on the diet have, however, failed to confirm this view. Constipation and diarrhœa are recorded as preceding the onset of an attack, and Crafts goes as far as to say that one or the other, generally the former, is an almost invariable pre-precursor in his patient. Other observers, however, have failed to observe this association.

The third factor, emotion, has only been recorded by Cousot, and is hardly noticed by other observers. It would, however, appear to have occasionally excited attacks in the case which forms the text of this paper.

Pathological Anatomy.

In the intervals between the attacks, all persons suffering from this disease have been practically healthy, and up to the present but little has been discovered abnormal during the periods of paralysis.

The *blood* has been examined by Goldflam, Taylor, Crafts and Mitchell, with almost entirely negative results.

The first three observers all record a slight relative lymphocytosis during the intervals, but the change is almost within the normal limits, and it is questionable if any significance can be attached to it. Examination of the blood cells in Mitchell's and my own case revealed nothing abnormal. A leucocytosis during an attack was found by Klein who examined one of Goldflam's patients, but this has not been confirmed by others. The toxicity of the blood serum was carefully estimated by Dr. Stengel in Mitchell's case, but found to be within normal limits.

Muscle.—Specimens have been examined by Goldflam and Crafts. The former describes a condition of increased diameter of individual fibres, together with some vacuolation, and to this he is inclined to ascribe the permanent electrical change he has found. Crafts describes much the same condition, and adds that there is a distinct increase in the amount of fibrous tissue; this finding is, however, discounted by the fact that he was using the gastrocnemius muscle. The vacuolation of the fibres is distinctly interesting when compared with the almost identical results obtained from the case described above; this change, which I believe to be artefact, would appear to indicate some abnormality in the muscle, rendering it more liable to shrinkage and alteration in being prepared for microscopic examination. When one considers the short duration of the paralysis, it seems hardly possible that any very gross changes in the muscle should be found.

Urine and fæces (by Dr. Goodbody).—The first reference of any importance as regards the examination of the urine was made by Goldflam. He estimated the toxicity of the urine by Bouchard's method, and found that, during the attacks, there was a distinct increase in the urotoxic coefficient, the increase, in some cases, being nearly fourfold. His first idea was that the poisonous substance was in the alloxuran group, but he afterwards came to the conclusion that this was not the case, as the results of analysis practically show equal amounts of this substance, both during the attacks and in the intervals. The final conclusion that he arrived at was that the active principle was of the nature of a toxic substance formed in the intestinal tract, but he made no attempt to isolate this substance, or substances, as described.

In Craft's report of the case investigated by him, there is no difference to be noticed in comparing the analysis of the urine voided during the attacks, and that passed during the interval. He found also an increased toxicity of the urine during the paralytic stage, but was unable to isolate the active toxic principle. Some experiments were carried out in connection with his case, in order to ascertain, if possible, whether any toxic principle could be isolated from the fæces. Extracts of the fæces were made in several different ways, and these extracts were injected into guinea-pigs, with the result that temporary paralysis, lasting about twenty-four hours, was observed in these animals which were inoculated with extracts of the fæces during the period of attack. In this case also, no success attended any attempt to isolate the toxic principle. So far, we have been unable to confirm his results on this subject.

Pathology.

The earliest opinion expressed as to the nature of the condition was that of Hartwig, who assumed that the paralysis was due to hyperæmia of the spinal cord, with temporary increase of serous exudation. Samuelsohn suggested that the paralysis was hysterical, but this may at once be excluded in the presence of such definite and characteristic evidence as the loss of reflex and electrical excitability. Westphal in 1885 was the first to suggest that the disease was due to some form of toxæmia, a view that was strongly upheld by Goldflam in 1890, who added that it was probably an auto-intoxication. This theory has been accepted by all observers since, the only variance of opinion being as to the *point d'appui* of the toxin in producing the palsy. Goldflam's own theory would place the lesion in the muscle fibres and motor nerve endings, a conclusion to which one seems forced by the electrical changes. The condition of the muscles with regard to electrical excitability is that of muscle which has gone beyond the stage of mere separation from its motor centre in the cord, such as is seen after recent section of the nerve fibre, or destruction of ant. cornual cells, and has arrived at a stage which corresponds with the functionally dead muscle seen in very old standing cases of infantile paralysis. This view is also strongly supported by the fact that the heart undoubtedly suffers, though to a less extent than the skeletal muscles, and even more so by the probability, as pointed out by Mitchell, that the muscle of the vessel walls suffers also. One point, however, which does appear difficult to explain, if we regard the paralysis as due to poisoning of the muscle fibres, and upon which very little stress has been laid, is the almost universal escape of the muscles supplied by the cranial nerves. There is no known anatomical difference that would account for their escape, and to suggest any selective action for a toxin, which otherwise acts so universally, is not in any way satisfying.

The only other notable theory, which was first enunciated by Cousot in 1887, and has recently been reformulated by Putman in 1900, suggests that the toxin, whatever it be, produces its effect by exciting the inhibitory action of cerebral or spinal centres. Such a suggestion it is impossible to disprove, seeing that we know little or nothing of the power that is called into action; further it is unnecessary as most of the phenomena are explicable without it.

One other important point remains to be considered, viz., as to the nature and origin of the toxin, the existence of which is practically admitted by all. Here we are verging into the realms of pure hypothesis, but certain points are worthy of discussion. In the first place two views are possible as to its nature and origin. (1) It is an entirely foreign substance, *i.e.*, a specific poison; (2) It is an increase in the amount of some of the toxins normally present in the body as a result of metabolism; the accumlation being due either to increased production or diminished elimination, or both.

With regard to the presence of a specific toxin, all that can be said, if we neglect Crafts' unsupported findings, is that all attempts to demonstrate such poison, either in the blood, urine or fæces, have signally failed. When, however, we come to consider the question of excess of what may be termed the normal toxins, we have some evidence of importance in that the urine shows an increased but otherwise unaltered toxicity.

The undoubted excitation of attacks by muscular fatigue recorded by so many observers here leads us to question whether the increased toxin is not that produced by muscular work. The result of severe exercise in an ordinary healthy person is a condition of loss of power in the fatigued muscles, which soon passes off. May it not be that family periodic paralysis is produced in this way in persons whose muscles are predisposed by heredity or otherwise to suffer more from the effects of such toxins. Having reached this point, we have still to explain the periodicity, and also the fact that attacks in the case described above were more frequent in bed than when taking exercise. The other factor in the accumulation of the poison in the blood is a diminution in the excretion and the suggestion may be made that for some reason, such as the complete rest is bed, excretion is not as complete as it should be. This suggestion would appear to be borne out by the results of diuretic treatment described above.

If these points are admitted it may next be asked why does the patient recover from the attack, seeing that the rest is still more complete and there is partial suppression of urine? The fact that urine voided immediately after an attack is increasedly toxic, and the copious sweating that is seen in many of the cases may serve to explain this difficulty, as we must assume that metabolism is at a very low ebb whilst the muscles are practically dead, and yet at the same time there is a slow excretion of toxin going on in the urine and sweat which would result in the course of time in a return to the normal condition.

A comparison with the disease described under the name of myasthenia gravis may render this hypothesis, viz., the accumulation of the products of metabolism of muscle more tenable, for there we find a condition in which muscle, as the result of work, loses both voluntary power and faradic excitability, recovering again more or less completely after rest. It must be pointed out, however, that in myasthenia gravis, although the faradic excitability may entirely disappear for the time being, yet the muscles still react readily to galvanic stimulation. This difference serves to clearly differentiate the two diseases, the explanation being probably that in family periodic paralysis the muscle fibres are affected, while in myasthenia it is the motor nerve endings that suffer; the clinical history of the two conditions is also quite distinct. Yet in spite of these differences there is a resemblance which will probably lead to the two diseases being classed together under the same clinical and pathological group.

Classification.

Until we know more of the pathology of the disease it is impossible to class it accurately with other diseases. The periodicity, together with the extraordinary change in the electrical reactions and reflexes, and the entire absence of all sensory symptoms, make the actual diagnosis easy. Goldflam, relying on the permanent electrical changes he has found, classes the disease with the myopathies. Taylor is more inclined to group it with the congenital myotonias. Both these views are probably correct in that there is almost certainly some hereditary, or at any rate congenital difference in the muscle fibres, causing them to suffer from the effects of the toxin; but the resemblance is closer, as pointed out above, to myasthenia gravis. The periodicity of the disease would also lead us with Mitchell to relate it to such conditions as migraine, in which it seems probable there is some form of periodic auto-intoxication, also markedly hereditary in character.

Prognosis.

The general opinion seems to be that the number of attacks tends to diminish towards middle age and sometimes they cease altogether. Goldflam mentions one case in which the attacks ceased at the first confinement. Life does not appear to be shortened by the disease, nor does death appear to have occurred as a result of the attacks, except possibly in the case of the father of Schachnowitsch's patient.

Treatment.

Has hitherto proved useless. Numerous drugs have been tried, such as quinine, bromide, arsenic, strychnine, &c., but all have failed. Electrical treatment, massage, &c., have also proved quite unavailing.

From my own observations, however, which require confirmation, I would recommend a regular, non-exciting life, with plain, simple food and moderate physical exercise. Diuresis should be promoted by the exhibition of large quantities of water, or as a pleasant and efficient substitute, imperial drink, while the bowels should not be allowed to become constipated. Turkish baths occasionally may be of value, but I cannot speak from experience. Drugs probably are not of much value, but if the attacks are frequent some digitalis and potassium acetate or citrate may be found useful.

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THE NERVE-CELLS OF THE HUMAN POSTERIOR ROOT GANGLIA AND THEIR CHANGES IN GENERAL PARALYSIS OF THE INSANE.

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ALTHOUGH there is a considerable amount of literature upon the changes in the cells of the posterior root ganglia in general paralysis of the insane, there still exists much diversity of opinion as to the extent to which those cells are affected, some authors describing advanced and destructive changes in the cells, while others are of the opinion that the affection is very slight.

It is worthy of note that before describing the changes to be found in general paralysis no one has attempted to give an account of the normal intimate structure of the posterior root ganglion cells; and it seems to us that a minute description of those cells, studied by the most modern methods now at our disposal, is an absolute necessity, because, as we hope to show, there are several types of cells which vary very markedly in size and in the arrangement of the chromophile elements, so that without a knowledge of the normal features one cannot attempt to describe the modifications which the cells undergo in the course of a disease such as general paralysis of the insane.

Early in our work it became evident to us that the older methods of fixation and hardening were utterly useless for the study of such a delicate cell as that contained in the spinal ganglia, and so we found it necessary to finally discard such fixatives as formalin and graduated alcohol.

As a result of adopting a new and reliable method of fixation, we have proved to our satisfaction that work conducted by the methods formerly used is full of artificially produced appearances, which in many cases have been mistaken for evidences of a morbid process. What those changes are we shall show before describing the changes found in the posterior root ganglia of general paralytics. But in the first place let us see how our knowledge stands with regard to the normal structure of the posterior root ganglion cells.

Before entering on a description of the types of cells found in the normal human posterior root ganglia we must refer to the work which has of late been done on the ganglia of the lower animals.

For several years it has been recognised that there were cells of different sizes in the posterior root ganglia, but, until comparatively recently, no attempt was made to classify the cells beyond the use of the terms "large" and "small." During the last five years, however, several observers have given some attention to the subject, and have classified the cells according to their size and to the arrangement of the chromophile elements within the cell.

Lugaro has summarised the literature on the subject and has given concisely the views held by various observers in his paper published last year; we shall refer to this fully later.

Dogiel, Ramon y Cajal and Oloriz recognised, when using Ehrlich's "*intra-vitam*" method, that there were large clear cells and small obscure cells.

Nissl, in 1897, described four types of cells in the posterior root ganglia of the rabbit; he distinguished them as follows:—

(1) Cells with large chromophile elements arranged concentrically around the nucleus.

(2) Cells having a single layer of elements arranged at the periphery, a thin clear zone just below this free from chromophile elements, and another clear space in the neighbourhood of the nucleus.

(3) Cells with a thin peripheral layer and a broader peri-

nuclear layer of large elements, and between these two a band of smaller elements, with sometimes some larger elements mixed with them.

(4) Cells somewhat similar to the preceding, but more or less indeterminate in character.

Van Gehuchten and Nelis, in 1898, distinguished the small and obscure cells from the large clear cells, and by the relative amounts of chromophile elements they distinguished numerous types which, they considered, were united by intermediate forms. They described the two types most commonly met with as being rich in chromophile elements, but in one these were large, irregular in shape, and scattered through the whole of the protoplasm of the cell; while, in the other variety, they were in the form of fine granulations uniformly distributed throughout the cell excepting a narrow clear peripheral zone.

Their third type included cells with a few fine irregularly shaped elements.

Next they described cells with large fusiform elements arranged concentrically around the nucleus.

They then mentioned a rare type in which a few large irregularly shaped chromophile elements were arranged exclusively around the nucleus; and, lastly, a still more rare type, whose elements were divided into two bands, one at the periphery and the other around the nucleus, and between the two there was a zone free from chromophile elements.

Cassirer in his description of the cells of the posterior root ganglia of the rabbit mentioned four types:—

(1) Large clear cells with the nucleus generally central, but sometimes displaced to the periphery; the elements in these were arranged around the nucleus, and at the periphery in concentric layers, and there was a narrow clear zone at the periphery; and a narrower one around the nucleus.

(2) Medium-sized cells, for the most part clear, with a large nucleus generally central, but sometimes excentric; around the nucleus there was first a zone of large chromophile elements, then one of very fine chromophile elements, and again at the periphery a zone of large elements.

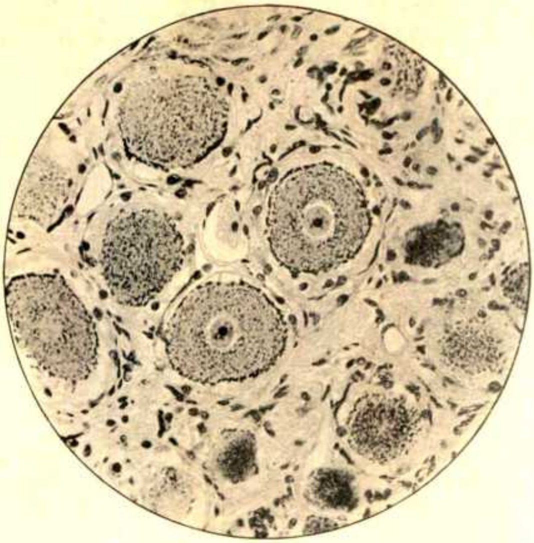

FIG. 1.

Two large clear cells, with central granuliform elements and a single peripheral ring of large chromophile elements. Note the perinuclear space. *Lugaro's type, No.* 2. Zeiss, apochrom., obj. 8 mm., oc. 4.

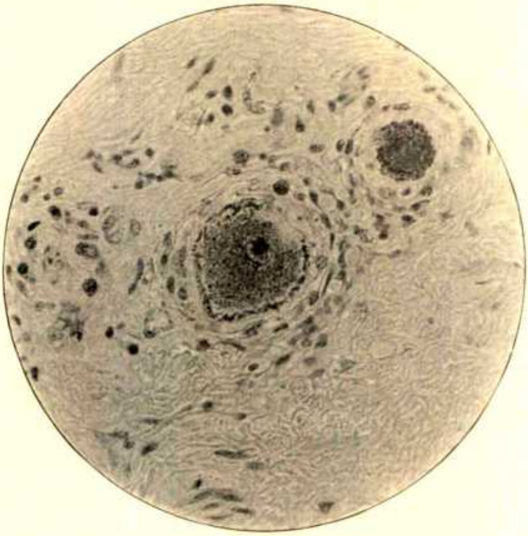

FIG. 2.

Early stages of chromatolysis in type 2. The nucleus is slightly excentric and the peripheral edge of the central granuliform mass is beginning to break up. Note the persistence of the peripheral chromophile ring. Zeiss, apochrom., obj. 8 mm., oc. 4.

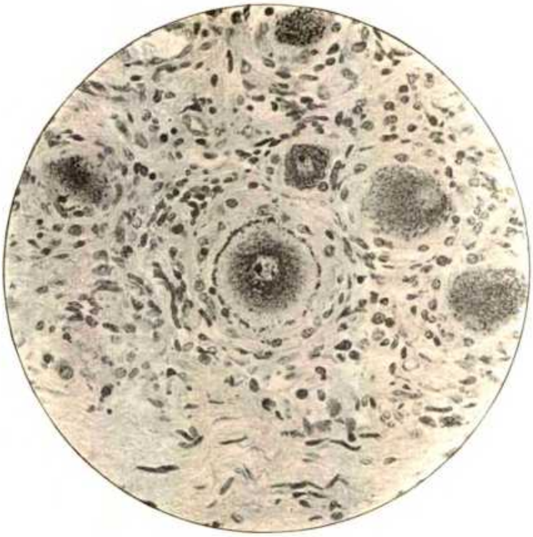

FIG. 3.

More advanced stage of chromatolysis in type 2. The central chromophile mass is very degenerated at its periphery and there is a clear space left between it and the large peripheric chromophile elements which are still well preserved. The nucleus here has retained its central position. Zeiss, apochrom., obj. 8 mm., oc. 4.

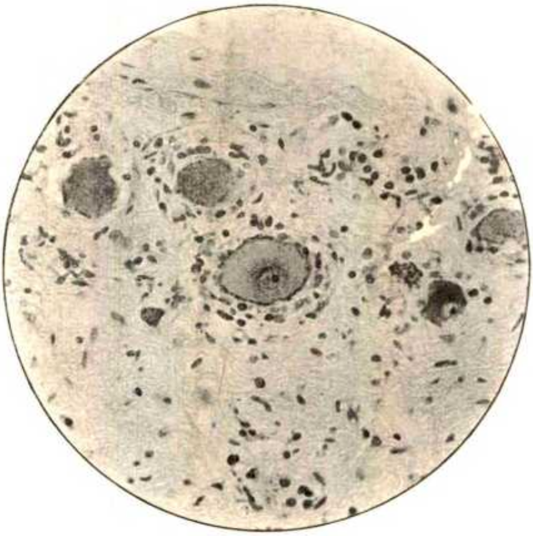

FIG. 4.

In this example of type 2 the central chromophile mass has disappeared with the exception of a few granules around the nucleus. Note that the peripheric chromophile ring still persists at this late stage of chromatolysis. There is a small dark-staining band of chromophile substance attached to the outer wall of the central side of the nucleus. Zeiss, apochrom., obj. 8 mm., oc. 4.

(3) Cells as large as the last, but of irregular shape, with a smaller nucleus, often excentric, and the elements indistinct because of the staining of the fundamental part of the cell.

(4) Small cells with a relatively large nucleus, with the elements arranged irregularly and with the fundamental part of the cell stained. Sometimes in these cells two nuclei were seen.

Cox also in 1898 published a paper in which he described the cells in the posterior root ganglia of the rabbit, but he differed from the above-mentioned authors in that he considered that the types of cells were clearly distinguished from each other, and he failed to find any intermediate forms between the various types.

He mentioned two types, and the first of these he divided into two sub-types, cells large and cells small.

In the large cells of type 1 the chromophile elements were not large, they were of irregular shape and only at the periphery did they assume an elongated form ; no concentric arrangement was seen excepting at the periphery and here there was only one layer so disposed. The nucleus was situated in the middle of the cell and was rarely double.

The small cells of type 1 were distinguished by the fact that the fundamental substance between the chromophile elements had a tendency to stain more or less deeply. The elements were distinct, but always much smaller than those in the larger cells.

The cells of type 2 had elongated chromophile elements arranged in layers, some layers were concentric around the nucleus, and others were concentric around the central point of the cell. The nucleus was always excentric, often markedly so.

He then referred to the different appearances which this type of cell presented in sections which were made in different planes, and showed that in one section the nucleus might be near the centre and in another placed at the extremity of the cell.

It will be seen that there are considerable differences between the descriptions of these cells given by the above mentioned authors; these differences have been cleared away, and in fact the classification of the types of cells met with in the posterior root ganglia has been placed on a firm and scientific basis by Lugaro in his valuable paper published last year.

Working with a new and improved technique, which will be described fully later in this paper, he divided the cells in the posterior root ganglia of the dog into five types, quite distinct from each other and having no intermediate forms. His first type consisted of clear cells, the largest in the ganglion, with fine granuliform chromophile elements scattered diffusely throughout the cytoplasm, and only a little increased in size and density at the periphery, internal to a narrow limiting clear zone, which was entirely deprived of them; around the nucleus also there was a narrow clear zone which was free from any chromophile elements; the nucleus was large and clear and was situated in the centre of the cell (fig. 6).

In his second type he included clear cells of medium size generally, although some of them equalled those of the first type. In these cells the chromophile elements around the nucleus were a little larger and less crowded than in type 1; they were bounded externally by a zone of varying width in which there were few, if any, elements, and outside this there was a single layer of large elements. At the periphery and around the nucleus there was a well marked narrow clear zone. The nucleus was large and clear and was always central (fig. 1).

The third type Lugaro spoke of as "scure" or darkly-staining cells. Many of these were small, but others were quite of medium size; they were frequently irregular in shape. The chromophile elements were very minute in the central part, and slightly larger towards the periphery. The distinguishing feature of this type was that the fundamental substance of the cells was stained more or less deeply, and this was the more marked in those of larger size. It gave rise to the dark, undefinable appearance similar to that seen in partially differentiated cells.

Frequently there was considerable difficulty in defining the outline of the chromophile elements, owing to the deep staining of the fundamental substance. The nucleus also was deeply stained; it was central, and was not relatively so large as in the other types. The peripheral and perinuclear zones were present, but were less distinct than in the clear cells.

The fourth type consisted of small or medium sized cells, in which the chromophile elements were large, few in number, and regular in shape, and had numerous branches attached; they were arranged concentrically around the nucleus, and there was a well marked peripheral and perinuclear clear zone. The nucleus was large, clear, and was situated centrally in the cell (fig. 8).

His fifth type of cell, corresponding to the type 2 of Cox, included cells of medium and small size. Some of these were round, and others were decidedly oval in shape. The nucleus was constantly excentric, often markedly so. The chromophile elements were arranged in concentric layers around the central point of the cell; those at the periphery were large, cohering, elongated, and united to each other by fine branches; those in the central part were smaller and more irregular, but the concentric arrangement was maintained. The peripheral and perinuclear clear zones were quite distinct (fig. 10).

The above description is a picture of a section of one of these cells made in the direction of the long axis of the chromophile elements; when the section was made in a plane at right angles to this, the chromophile elements were seen to have an irregular shape, and they were connected by their branches so as to present a reticulated appearance; their size, the excentricity of the nucleus, and the concentric arrangement of the chromophile elements will assist one to recognise them.

Moreover, intermediate forms, in which the section was made obliquely to the long axis of the chromophile elements, were frequently seen. In some cells too, the nucleus was placed at one end of the cell, with layers of chromophile elements disposed concentrically around it, and at the other pole of the cell there was a distinct spiral arrangement of the elements.

This cell clearly corresponds to the cell referred to by Lenhossek as being found in the frog, in which he spoke of this disposition of the nucleus and spiral as "centrosome" and "sphere," and he considered it a cell in process of reproduction.

Levi, however, in 1897, noticed a similar cell in the toad, and he showed that in this type of cell the nucleus, with concentric layers of chromophile elements around it, was normally situated at one pole of the cell, and that the other pole was occupied by a figure, which he described as a "spiral vortex of fibrils and chromophile elements."

The above are the five types of cells which Lugaro described in the posterior root ganglia of the dog.

In his article on the posterior root ganglia of the rabbit he included types 1 and 2 under one head, because he found that in the normal cells there were some points of similarity; and he also found that, in the course of their degeneration after section of the afferent nerve, they passed through the same definite series of changes, and only at the very commencement were they distinguishable.

At the beginning of our work on this subject, we examined the posterior root ganglia of lower animals with the view of confirming the findings of Lugaro, and we then directed our attention to the root ganglia in man.

We have cut sections of the posterior root ganglia of the cat, the dog, and the rabbit, and we can fully confirm the description of the types of cells given by Lugaro; and a careful examination of the root ganglia in man has shown that the same types of cells exist there also.

We have followed Lugaro's classification, because it is the most recent and the most complete, and because we consider that a comparison with all the descriptions given above might lead to some confusion.

Our normal specimens were obtained from cases of acute mania who died soon after admission into the asylum. We feared that it would be impossible to eliminate the fallacies due to imperfect fixation and to *post-mortem* change, if we relied on tissues obtained from a general hospital.

CELL TYPES IN THE NORMAL HUMAN POSTERIOR ROOT GANGLIA.

In the human posterior root ganglia we have been able to separate and to distinguish five independent varieties of cells, which show differences both in the normal arrangement of the chromophile elements, and in the way in which they undergo modifications in their degenerative phases.

The most common type of cell met with is that which corresponds to Lugaro's first type. The cells in this type are large and clear; the chromophile elements are granuliform, and are scattered throughout the entire cell body; but at the periphery there is a slight increase in their size (fig. 6). Outside the chromophile elements there is a well marked clear peripheral zone devoid of chromophile substance, and around the nucleus which is central, there is also a similar well defined clear zone, called the "perinuclear space."

The next type in frequency is that which corresponds to Lugaro's second type in the dog. We find this cell is clearly distinguishable from the preceding by the large size of the peripheral chromophile elements, which are arranged in a single ring around the cell (fig. 1). Internal to this ring the chromophile material is granuliform, the individual elements being very minute and irregular in size.

The granules are usually slightly increased in number around the centrally placed nucleus.

The peripheral and perinuclear clear zones are again prominent features, as in the first type.

The third and last type of cell, in which the chromophile material is exhibited in the form of granules, is that termed by Lugaro the "scure" or darkly-staining cell. In the human subject we find a large and small variety of this type.

In the smaller cells the chromophile elements are very minute, with indistinct outline, and are scattered diffusely throughout the cytoplasm. In the larger cells the chromophile elements are larger, and are arranged in two concentric condensations—one around the nucleus and the other towards the periphery. In both the fundamental amorphous substance stains deeply, and obscures to a large extent the chromophile material; the nucleus, which also stains deeply, is central. A perinuclear clear space is recognised with difficulty, but the peripheral clear zone can always be seen, though narrower than in the large clear cells.

The two remaining types, about to be described, are sharply defined from the preceding three varieties by the large size of the individual chromophile elements. The granuliform chromophile substance, which forms the main feature in types 1, 2, and 3, is replaced in types 4 and 5 by an element which is much larger and more distinct, but the shape and arrangement is not the same in the two types.

Under type 4 we find two varieties, one with small cells and the other with cells of medium size, and in these there are certain characteristics common to both. The chromophile elements are large and comparatively few in number; the fundamental achromatic substance is either unstained or only very slightly coloured; the nucleus is central, and the peripheral and perinuclear clear spaces, which we have mentioned as being common to all the types, are prominent features in these cells (fig. 8).

In the smaller variety the chromophile elements are smaller and a little more slender than in the medium-sized cells, and they are arranged concentrically around the nucleus.

In the medium-sized cells the chromophile elements are larger, and tend to assume a diamond or square shape, and they do not present such a complete concentric disposition as is seen in the smaller variety. Both varieties, therefore, of this type can be clearly differentiated from any of the preceding types by the large volume and the well-defined outline of the chromophile elements.

In the other type of cell, which corresponds to Lugaro's fifth type in the dog, the chromophile elements are as large as in type 4, but differ from them in their shape and in their disposition around the nucleus. The nucleus is normally markedly excentric and frequently occupies a peripheral position, but it is always separated from the extreme edge of the cell by a narrow band of chromophile substance. The chromophile elements are long and slender in the large examples of this type (fig. 10), and they radiate from the nucleus in successive rings towards the opposite pole of the cell. The elements are more closely packed together around the nucleus, but further away the space between adjacent rings is greater. In the smaller vorticose cells the elements are a little thicker, relatively to their length, than those in the larger variety, but the same arrangement in the cell prevails.

Sections of these cells made in different planes exhibit the same pictures as those described by Lugaro in the cells of the dog.

Various authors who have written on the subject of the posterior root ganglia have drawn attention to the fact that the size of the cells varies considerably in the ganglia of the different regions of the cord, the great majority of cells in some ganglia being large and in other ganglia small; and they have suggested that as the cells differ in size in various ganglia, their function also must be distinct. We have found that the cells do vary in size in different ganglia, but in each ganglion every type of cell is present, and there can be no doubt that in the ganglia in which the cells generally are small, those cells which belong to the smaller types are reduced in size, as well as those which belong to the larger types, and we must assume that the same type of cell has the same function in every case. It is hardly correct, therefore, to say that the large cells are absent or relatively greatly reduced in number in certain ganglia.

Another source of fallacy lies in the fact that in certain ganglia almost all the cells along one side may be of large size. If a section was made along this side, practically only large cells would be seen; but if a section was made at right angles to this, the various types would be seen in their usual proportions. We have, therefore, found it necessary to cut sections right through the ganglion.

THE PERINUCLEAR SPACE.

In the above description of the cell types, reference has been made to a perinuclear space, which forms one of the constant histological appearances met with in the nerve-cells of the posterior root ganglia.

This perinuclear space is worthy of special mention, in view of the importance now attached to the intracellular lymphatic system and its connections, as there seems to be a large amount of evidence to show that this space forms part of the canalicular system described by Donaggio, Holmgren, and others.

It is not our intention to enter into a description of the lymphatic canalicular system or of the endocellular vessels (Fritsch, Holmgren, and Adamkiewicz), as such is quite outside the scope of this paper; but we may be pardoned for dwelling on the perinuclear space a little more fully, as it might be questioned whether the appearance was really a normal feature of the cell, or an artefact due to the effects of hardening or shrinkage.

Below it will be seen that considerable trouble has been taken to select the best fixative for these delicate structures in order to eliminate as far as possible faulty fixation or shrinkage during the subsequent operations of hardening and embedding. But first let us look at the opinions to-day advanced as to the precise nature of this structure.

In connection with their work on the endocellular lymphatic canalicular system, Holmgren and Donaggio have observed and described a distinct perinuclear space, containing no chromophile material, into which the canaliculi appear to open, and Holmgren seems to consider that the perinuclear space is to be regarded as the centre of the endocellular circulatory paths. We have ourselves observed two or three of these canaliculi in some cells of the first type, but we have not yet seen any instances where they open into the perinuclear clear space. The canaliculi are seen as clear, sinuous bands, surrounded on both sides by chromophile elements. Confirmatory evidence of the existence of this structure is to be

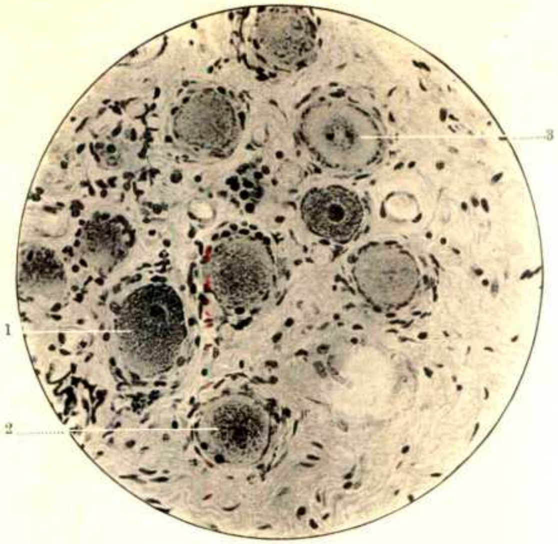

FIG. 5.

Note three cells of type 2 showing—(1) Early chromatolysis with excentricity of nucleus; (2) Degeneration of periphery of central granuliform mass; (3) Disappearance of central chromophile elements and persistence of peripheric chromophile zone. Zeiss, apochrom., obj. 8 mm., oc. 4.

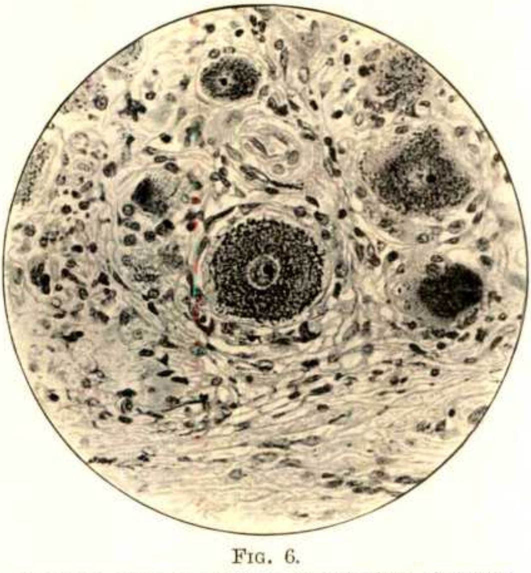

FIG. 6.

A large clear cell with granuliform chromophile elements. There is a slight increase in size of the elements towards the periphery but no definite prominent peripheric ring as in type 2. *This is Lugaro's 1st type.* Note the perinuclear and peripheral clear spaces. Zeiss, apochrom., obj. 8 mm., oc. 4.

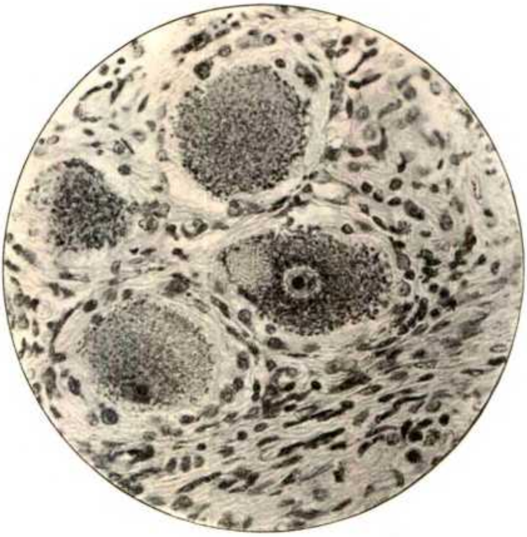

FIG. 7.

Two large clear cells of type 1. In one there is a small localised deposit of pigment at one extremity of the cell; in the other there is a diffuse chromatolysis with marked excentricity of the nucleus. Zeiss, apochrom., obj. 8 mm., oc. 4.

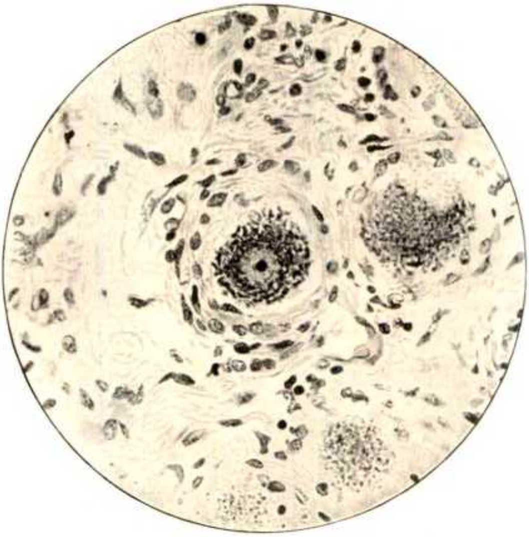

FIG. 8.

Medium-sized cell with large chromophile elements. Note the difference in size between the elements in this type and types 1 and 2. *This is Lugaro's 4th type.* Leitz, obj. 7, oc. 1.

found in the embryological researches of Fragnito, Colucci, Capobianco, and Piccinino, who, in working at the development of the nerve-cell, lay special emphasis upon the peculiar mode of development of the perinuclear space and the canaliculi.

Without going completely into a consideration of these authors' work, it is sufficient for our present purpose to note that they demonstrate the pluricellular origin of the nerve-cell, and in the course of their work they show how in the process of development the perinuclear space is formed, and that it is present at an early period of the existence of the cell.

The above-mentioned authors are agreed that the nerve-cell is formed from a primary neuroblast and several secondary neuroblasts, and that each neuroblast is surrounded by protoplasm.

The primary neuroblast becomes the permanent nucleus of the adult cell, while the latter become associated with it and undergo certain modifications to form the adult cell-body with its contents. Fragnito then shows that between the primary neuroblast and the secondary neuroblasts there is a space, which becomes the perinuclear space, and that this communicates with the spaces between the secondary neuroblasts, which become the canaliculi of the adult cell. In support of Fragnito's observations, Colucci affirms that the protoplasmic substance surrounding the primary neuroblast presents no true continuity with the nuclear wall. There is here a clear interposing ring in which there is a very faint, refractile, substance. As the surrounding protoplasm grows, the more evident does this clear ring become.

There can be little doubt, then, that the perinuclear ring is not an artificially produced appearance. It may be suggested that the reagents used in fixation and hardening caused retraction of the chromophile substance from the nuclear wall: but the observations of Colucci were made on fresh as well as hardened nervous tissues, stained with aniline dyes, and he found the same appearance in both instances.

Peripheral Clear Ring.

There remains yet to be noted the peripheral clear ring, which also forms one of the histological features of all the cells types mentioned above. This ring varies in width according to the type of cell, being larger in the large cells and smaller in the small cells.

The chromophile elements do not reach the margin of the cell body, but terminate abruptly, leaving a clear zone around the entire cell. This is especially well seen in types 1 and 2. The outer margin of this zone is regular in outline and stains slightly more deeply, and so gives to the cell a well-defined edge and separates it sharply from the surrounding capsule.

In this peripheral clear zone no trace of chromophile substance can be seen, but minute fibrils are observed, with high magnification, to traverse it in all directions, giving one the impression of a fibrillar network. Between the external margin of the peripheral clear protoplasmic ring and the capsule enclosing the nerve-cell, there is a narrow pericellular space, crossing which one observes numerous minute, faintly-staining threads, which apparently run from the surrounding tissues into the peripheral clear zone of the cell body.

As to what these thread-like structures are, we hesitate to give a decided opinion; but the observations of Holmgren, Studnicka, Donaggio, and others, lead us to suggest that they may be of the nature of vascular or lymphatic structures.

Seeing that we have not employed any special technique or staining to warrant us in giving any definite view on this subject, we at present merely make a statement of their existence, while describing the peripheral clear ring which surrounds the ganglion cell.

Fixation, Hardening and Embedding.

The choice of a suitable fixative and the subsequent hardening in alcohol are of the utmost importance in dealing with such delicate structures as the ganglion cells under consideration.

It is interesting to note first the researches of Lugaro in this connection, and his results.

The following solutions were employed by him :—

(1) Equal parts of a saturated solution of corrosive sublimate and picric acid.

(2) Mann's solution (sublimate solution, picric acid and formaldehyde).

(3) Carnoy's fluid.

(4) Gilson's fluid.

(5) Nitric acid (5 per cent.).

(6) Cox's first mixture (saturated sublimate solution, 30 parts; osmic acid, 1 per cent., 10 parts; acetic acid, 5 parts).

(7) Cox's third mixture (saturated sublimate solution, 30 parts; formol, 10 parts; acetic acid, 5 parts).

The results were as follows :—In Cox's fluids some shrinkage occurred, while Carnoy's fluid fixes the centre better than the periphery of the cell. The mixture of saturated sublimate solution and picric acid, Mann's fluid and nitric acid gave the best results, not a single cell showing the least appearance of shrinkage.

In staining, Lugaro found toluidin blue the best stain, and in the process of differentiation he recommended absolute alcohol in preference to any essential oil. An accessory stain, such as erythrosin, he found to be harmful in its action, because it took place at the expense of the chromophile elements, the smallest of which tended to disappear. The selective properties of the stain differed with the fixing agent; with picric acid and sublimate and with Mann's fluid the smallest chromophile elements were projected with incomparable clearness on a transparent background, with nitric acid the colour was paler and the contour of the chromophile elements less distinct. Carnoy's and Gilson's fluids gave good results, but they were inferior to the others named. Delafield's hæmatoxylin gave good results after fixation in picric acid and saturated sublimate solution, and in Mann's fluid. We find that it is necessary to use this stain in dilute solution (1-80), and good results are obtained after one hour's immersion.

In our own work we have used as fixatives,

(1) Mann's fluid.
(2) Saturated sublimate solution (Heidenhain).
(3) Saturated sublimate solution and picric acid.
(4) Nitric acid, 5 per cent.

We have found that the mixture of saturated sublimate solution and picric acid in equal parts gives us the best results with toluidin blue staining, although it may be mentioned that saturated sublimate solution alone is also a very good fixative.

It is impossible to attach too much importance to the special fixation and embedding methods which must be employed for the cells of the posterior root ganglion, because without such special fixation and staining, a recognition of the varieties or types of cells which comprise the ganglion is quite impossible.

The mode of preparation of the human posterior root ganglion, which we have finally adopted, after a somewhat lengthy trial, is as follows:—The ganglia to be studied must be obtained from the cadaver before *post-mortem* changes have had time to affect the cells even in the slightest degree. It can easily be understood that no matter how good the fixative employed may be, if *post-mortem* changes have set in an accurate picture of the nerve-cells cannot be obtained, and it can readily be demonstrated that the earlier the tissue is fixed the more do the results approximate to the appearances observed in animals whose ganglia have been fixed immediately after death.

The ganglion, which is carefully separated from the cord, is split into two pieces with a sharp razor, as we find that, without such an incision, penetration by the fixative is greatly delayed on account of the density of the capsule surrounding the ganglion and nerves. The division of these structures materially facilitates the rapid fixation which is so necessary, and allows the sublimate and picric acid to penetrate the ganglion from both periphery and centre.

The tissue is allowed to lie in the solution for three days—a less time scarcely suffices for the fixation of the larger ganglia; at the end of this time the fixative is poured off, and the process of hardening in graduated alcohol, commencing with 40 per cent., begins. When the hardening is completed the tissue is at once embedded in celloidin in the usual way, because we consider it preferable to keep the tissues in celloidin rather than to allow them to undergo prolonged immersion in alcohol. When required for sectioning, the embedding is completed by immersion in origanum oil and paraffin (equal parts) for twelve hours at a temperature of 45° to 50° C.; finally in paraffin alone for twelve hours, care being taken to reduce the deleterious effects of over-heating in the bath to a minimum.

The combined method of celloidin and paraffin embedding has been employed because it serves the double purpose of retaining the cells in position and at the same time allows of the thinnest possible sections.

It is necessary, in our opinion, to use celloidin as otherwise the cells may fall out of the section, leaving empty spaces, an appearance which will be referred to later.

We have laid special stress on the above technique because it seems to us that faulty fixation is entirely responsible for the spaces and shrunken cells, often described as signs of morbidity. Such so-called morbid appearances are very easily obtained in the posterior root ganglia of healthy lower animals, if one allows *post-mortem* change to become established, or if an inadequate fixative, such as alcohol, is alone used.

Tissues from the healthy animal have been treated by the various solutions mentioned above, as well as morbid tissues from the insane, and whereas the healthy cells, where faulty fixatives were used, exhibited a considerable degree of shrinkage, the cells in the morbid ganglia, treated by the most recent methods of technique, showed no sign of such an appearance.

When shrinkage of the nervous tissues does occur, whether from imperfect fixation, *post-mortem* change, or both combined, the appearances obtained are constant and typical, and, as will be shown later, do not resemble in the slighest degree the pictures of true chromatolysis.

The first portion of the cell to be affected by the shrinking process is the peripheral clear zone previously mentioned. The pale achromatic substance forming this structure first loses its definite outline and deep indentations appear on its edge; as the change progresses, these indentations increase, until they reach the external limits of the chromophile part of the cell. On this stage being reached the cell is found to be without a proper peripheral clear ring, and this structure persists only in thick and thin deep staining bands uniting cell with capsule, and bridging across the artifically enlarged pericellular space.

In our description of the normal clear peripheral zone, it will be remembered that we described fine pale threads passing across the pericellular lymph space, and forming the only evidence of anatomical continuity between the nerve-cell and its capsule. It seems to us that it is along those threads that the cytoplasm shrinks, collecting on them, and thus giving rise to the thickened bands attached to the tissue surrounding the cell; it is only in this manner that we can see our way to account for the disappearance of the pale clear peripheral zone, which we know exists in the normal unshrunken cell, and for the development of the bands which occupy the former position of the peripheral protoplasmic ring. In consequence of this shrinkage the staining processes also are very much interfered with, the elements take the dye in a different manner, and the recognition of the cell types becomes a matter of no small difficulty.

With a more severe degree of shrinkage the bands of altered cytoplasm are prone to rupture, and on manipulation of the section, the cell drops out of its capsule leaving a clear open space. Such an appearance we have not yet seen as a result of a true degenerative process, and we hope to show that as the cell undergoes disintegration, its site is gradually filled up by proliferation of the surrounding connective tissue nuclei.

It seems to us therefore that unless *post-mortem* change and artifical shrinkage are entirely eliminated, the existing true pathological processes are to a very large extent masked, and artefacts or pseudo-pathological processes receive an attention sufficiently great to lead to a grave source of fallacy in the estimation of the extent of the morbid change present.

MORBID CHANGES IN THE CELLS OF THE POSTERIOR ROOT GANGLIA IN GENERAL PARALYTICS.

It has been necessary to enter into a lengthy description of the cell types in the normal posterior root ganglia, for the reason, that each type degenerates in a specific manner distinct from the others, and a knowledge of their normal structure is therefore necessary, in order to recognise each type at an early stage of degeneration, and to follow it through its progressive phases. This we have done in six typical cases of general paralysis, uncomplicated by tabes, only examining such ganglia as we were able to secure before *post-mortem* change had set in. In every case ganglia were taken from the cervical, dorsal and lumbar regions, and having been prepared according to the method recommended above, were stained by Lugaro's modification of Nissl's method. Toluidin blue being used in preference to thionin.

The chromatolysis revealed by this method must naturally be described under each of the five types of cells existing in the ganglia.

Of the two varieties of large, clear cells which are described as No. 1 and No. 2, the former persists practically unaltered, much more frequently than the latter. Chromatolysis is shown in the first type by a general breaking up of the granuliform elements throughout the cell, and by an early peripheral displacement of the nucleus. The elements around the nucleus often show a tendency to preserve their shape for a considerable time after those in the other parts of the cell have disintegrated. A cell in an advanced stage of degeneration shows its altered chromophile elements in the form of a fine dust scattered throughout the cell body (fig. 7).

The second type of cell, though structurally somewhat closely resembling the first type, is easily identified owing to the characteristic peripheral chromophile band which persists to a very late period of degeneration. The seat of the primary stage of degeneration is much more local than in the preceding type.

It will be remembered that the centre of the cell body is occupied by a granuliform mass of elements, and it is at the periphery of this mass, that is, immediately internal to the peripheral chromophile ring, that we have observed the process to begin (fig. 2).

The disintegration proceeds progressively towards the nucleus, and at the same time the peripheral large chromophile elements are seen to become united by their extremities to form a complete ring around the cell (fig. 3). With the progression of the degeneration, the central granuliform mass gradually disappears, until in the later stages the cell is seen to possess a well-stained nucleus and a peripheral chromophile ring only, the two being separated by clear, unstained cytoplasm (fig. 4). Occasionally, when the chromatolysis has reached this stage, a small, diffuse, chromophile mass is seen attached to the nuclear wall (fig. 4), and when the nucleus is excentric, the mass is present only on the side of the nucleus which is turned towards the centre of the cell.

During the progress of the chromatolysis the nucleus may pass to the periphery of the cell, but very frequently it remains central or nearly so.

In the "scure" cells (type 3), with the onset of chromatolysis, the nucleus passes towards the periphery and the centre of the cell becomes clearer owing to the breaking up of the chromophile elements. The degeneration spreads until chromophile elements are recognisable only at the periphery, and the remainder of the cell stains diffusely blue.

The cells at this stage somewhat resemble the cells of type 2 in an advanced stage of degeneration, but they are much smaller in size, and do not stand out so clearly from surrounding structures, and the nucleus is much more

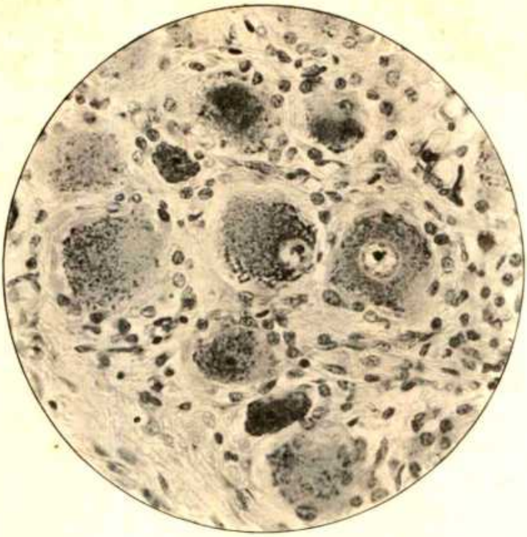

FIG. 9.

Note the cell in centre of the field. The nucleus is excentric and in its vicinity the chromophile elements are large. At the opposite end of the cell the chromophile material is broken up into fine granules. This is a degenerating No. 4. Zeiss, apochrom., obj. 8 mm., oc. 4.

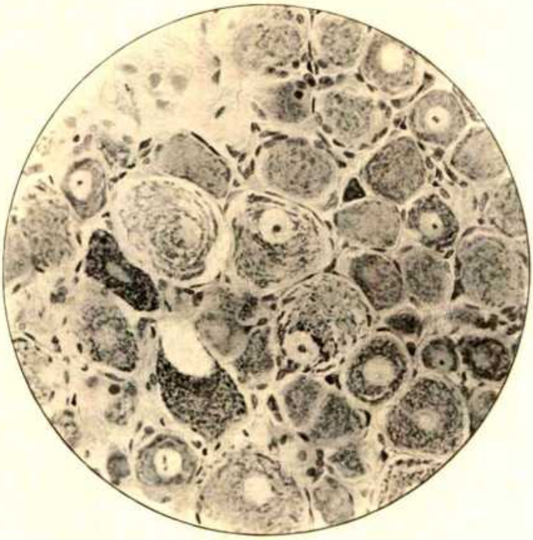

FIG. 10.

Two cells in centre of the field with elongated chromophile elements and excentric nucleus. Those are Lugaro's 5th type, and called by him "the vorticose cell." Zeiss, apochrom., obj. 8 mm., oc. 4.

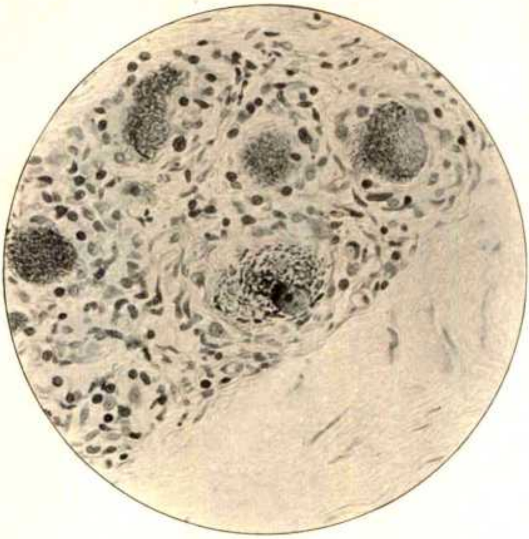

FIG. 11.

Note cell with excentric nucleus, attached to which there is a chromophile condensation which points towards the centre of the cell. At the opposite pole of the cell the chromophile elements are breaking up. This is a degenerating vorticose cell. Zeiss, apochrom., obj. 8 mm., oc. 4.

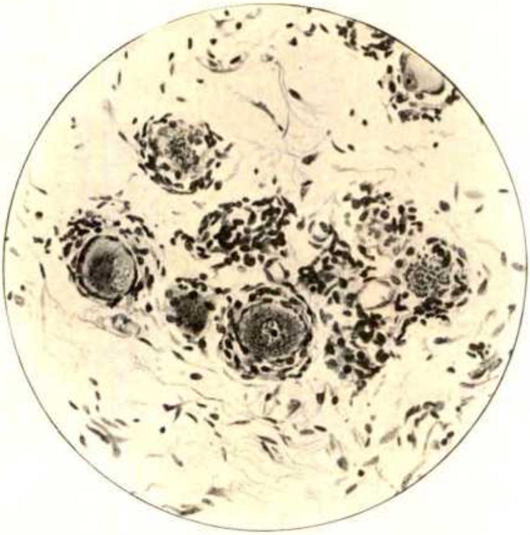

FIG. 12.

There are two degenerating cells around which can be seen a proliferation of nuclei. Above those two cells aggregations of nuclei are seen occupying the former site of degenerated nerve-cells. Zeiss, apochrom., obj. 8 mm., oc. 4.

frequently displaced to the periphery than in the large clear cells.

In type No. 4 the chromatolysis may commence at one focus in the cell, or may have a diffuse character from the beginning. The large elements are seen to become broken up into small, irregular granules, and the nucleus tends, at an early stage, to assume an excentric position, and when it has reached a point quite close to the periphery the largest remaining elements are observed in its immediate vicinity (fig. 9), while those in the other parts of the cell are represented by a fine diffusely staining dust.

This type of cell is always recognisable in its later phases of degeneration by the prominent, thick, chromophile elements placed between the sides of the excentric nucleus and the edge of the cell (fig. 9).

It has been difficult in the last type, that is the vorticose cell, to follow out the morbid changes, because of its comparative rarity. We have examined numerous sections from the ganglia of general paralytics, and from cases of acute insanity, and we have been struck by the scarcity of this type of cell. Dr. Eades, who is at present working at the changes in the posterior root ganglia of epileptics, confirms this observation, having himself noticed how infrequently this type of cell is met with. We have, however, discovered cells in various phases of degeneration, and in them we have noticed that the chromatolysis commences at the periphery by a breaking up of the elements, and at the same time there is frequently a large condensation of deep staining chromophile substance attached to the side of the nucleus which is turned towards the centre of the cell (fig. 11).

The nucleus maintains its peripheral position, and at its sides some slender, elongated elements remain practically uninjured. These elements are no longer seen in the other parts of the cell. The elements which do remain intact are never so thick or so prominent as those seen in the cells of type 4 at a similar stage of degeneration, so that an examination of the elements around the excentric nucleus will still reveal the type to which the cell belongs.

Although it has been possible to classify the stages of chromatolysis of the various ganglion cells under the above-mentioned types, we have seen a few cells which have become so disintegrated as to be beyond such classification.

When cells reach a more advanced stage of degeneration than that described above, we have noticed that the surrounding connective tissue nuclei proliferate and form a dense ring around the cell residue, encroach upon it, and gradually fill up the site previously occupied by it (fig. 12). Such extensive forms of degeneration, with nuclear proliferation around, occur with great infrequency, and it is only around cells showing this advanced stage of disintegration that any degree of nuclear proliferation is found, and moreover, it progesses *pari passu* with the destruction of the cell, until finally there is only a group of proliferated nuclei left to indicate the position which the ganglion cell originally occupied (fig. 12).

Notwithstanding the fact that so many ganglion cells show a considerable degree of morbid change, yet it would be quite erroneous to assume that those cells were dead, seeing that their nuclei not only maintain their regular outline, but also show a normal affinity for the staining reagent. Such being the case, we regard them as still capable of regeneration, a fact which is borne out by the experimental work of Lugaro and others.

His work on the cells of the posterior root ganglia in the dog and rabbit after resection of the afferent nerve proves conclusively that, although the chromophile elements may almost entirely disappear, yet complete restoration is possible, and the normal appearance of the cell may be re-established on the completion of the process of repair.

In our sections we have frequently noticed appearances which suggest an attempt at regeneration of the chromophile material taking place in the cell.

When the breaking up of the chromophile elements has reached its height, we have found not infrequently a small chromophile mass attached to the outer side of the nuclear wall which looks towards the centre of the cell, and in examples which may be taken to show more advanced stages of regeneration, this mass may reach a considerable volume, occupying a large part of the centre of the cell, and Lugaro has shown that it is from this chromophile mass that the new Nissl-bodies are formed by diffusion and reconstruction of the chromophile granules.

Such appearances, coupled with the fact that the nucleus of the nerve cells invariably remains well stained, and persists in an apparently healthy condition until the latest stages of chromatolysis seems to explain to us why there are so many cells found showing the various stages of degeneration which we have described, and why there are so few cells which go on to complete destruction.

Granted that the nucleus does not die, then regeneration is possible on the removal of the cause of the morbid change; and it can hardly be doubted that the cause in this instance is the presence of toxines, which are now admitted by all to be present in the blood of general paralytics.

But it is conceivable that the toxicity which affects the tissues in this disease, is not constant in its intensity, but is liable to waves of remission or intermission during which the affected tissues, if not too severely injured, can recover, and this fact is borne out by clinical evidence.

It can easily be understood, then, how it is that we have found so few examples of the destruction of cells in the posterior root ganglia of general paralytics, and we believe that the cells of this system become affected by the action of toxines upon them, but that on the removal of those toxines, the cells are capable of regeneration, and that probably they may react to the poisonous effects of those toxines and become repaired more than once in the course of the disease, before their resistive power is finally overcome, and they undergo complete destruction.

We have shown that many cells become chromatolysed in consequence of the action of toxines upon them, but the chromatolysis stops short of actual destruction of the cell, leaving the nucleus apparently unimplicated. Recovery being still possible, it can readily be understood that the central prolongation of those cells ought not to undergo a secondary degeneration; and as a matter of fact repeated examinations show that they do not. The posterior nerve roots examined by the Marchi and Weigert-Pal methods, even in teased preparations, present a very slight degree of degenerative change, and this is not to be wondered at, when one considers how few ganglion cells show extreme phases of chromatolysis such as would lead one to suppose that they were dead.

In this connection one cannot help noticing how frequently one finds an amount of fibre degeneration in the posterior columns of the cord, which cannot be accounted for by the degree of degenerative change in the posterior root ganglia. We acknowledge that here we are at variance with Sibelius and Vyrubow, who affirm that the degeneration in the posterior columns depends on the destruction of the posterior root ganglion cells. A glance at their description of the alleged morbid appliances reveals the fallacies contained therein, as they base their conclusions on such descriptions as a granuliform degeneration of the chromophile elements—although as we have pointed out a granuliform condition of the chromophile elements is a normal feature of types 1, 2, and 3—and a paucity of cells revealed by the fact that there are clear spaces in which no cell structure can be detected. Such pictures as the latter can never be accepted as indices of morbidity, as we have demonstrated that *post-mortem* change and faulty fixation can account for them entirely.

In addition to this, experience has shown us that as the cell goes on to complete destruction, its site becomes gradually occupied by proliferation of connective tissue nuclei. Even in acute insanities, where one finds a much more rapid form of chromatolysis in certain cells, although the chromophile substance may be wanting almost throughout the entire cell, yet the cell retains its normal shape and preserves its proper relationship with the surrounding structures.

As we have indicated before, spaces and shrinkage are artefacts, and if discounted from Sibelius and Vyrubow's work, the amount of fibre degeneration will assume a preponderance greatly in excess of the true degenerative change in the ganglia, and the results of those authors' research will more nearly approximate to those found by us.

We are not alone in considering that the changes in the posterior root ganglia are insufficient to account for the changes in the posterior columns, as Campbell and Fürstner have found little abnormality in the ganglion cells beyond a tendency to hyperpigmentation. Rubaud supports the above view, and considers that the scattered degeneration of the posterior columns occurs secondarily to an affection of the cells in Clarke's column. It seems to us more likely, however, that the diffuse degeneration found in the posterior columns in general paralysis uncomplicated by true tabes, is due to the toxines in the circulation which are capable of attacking the fibres everywhere in their course throughout the cord.

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ON THE CHANGES IN THE NERVOUS SYSTEM IN A CASE OF OLD-STANDING AMPUTATION.

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HISTORICAL.

FOLLOWING amputation of a limb, hemiatrophy of the spinal cord was observed by Bérard[1] in 1829. Krause and Friedländer[2] found that both gray and white matter in the spinal cord underwent atrophy on the same side as the amputation. Vulpian, Dejerine-Mayor, Hayem-Gilbert, Edinger, Gudden, Erlitzky, Homén and Vanlair confirmed this statement as regards the gray matter. Vulpian[3] noticed atrophy of the posterior horn alone on the side of amputation; Hayem-Genzmer and also Vanlair[4] found the anterior horn alone affected; while Hayem-Gilbert, Drechfeld, Edinger, Krause-Friedländer, Homén[5] and Bignani and Guanieri[6] state that both anterior and posterior horns are affected.

Varying statements are made as to the condition of the cell-groups in the anterior horns on the side of amputation (or nerve division). Thus Dejerine-Mayor[7] state that the antero-internal group exhibit a defect in number of cells; Drechfeld, Hayem-Gilbert,[8] and Homén on the other hand, find that the postero-lateral group is chiefly but not exclusively affected; while Krause and Friedlander find that the postero-lateral group is alone affected. According to the last two observers, and also Homén, Clarke's column is affected as well.

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[2] "Ueber Veranderungen der Nerven und des Rückenmarkes nach Amputationen." *Fortschritte der Medicin*, 1886, S. 749.

[3] *Archives de physiol. norm. et pathologique*, 1868, I., p. 443; 1869, II., p. 675.

[4] "Des altérations nerveuses centripètes consécutives à la section des nerfs, etc." *Académie Royale de Médecine de Belgique*, 1891.

[5] "Veränderungen des Nervensystems nach Amputation." *Beiträge zur pathologischen Anatomie von Ziegler*, 1890, S. 304.

[6] *Bolletino della R. Academia di Roma*, 1888.

[7] *Bulletin de la Société de Biologie*, 1878.

[8] *Archives de physiol. norm. et path.*, 1884, III., p. 480.

By Hayem-Gilbert a diminution in size of the nerve-cells of the spinal cord is described; Genzmer, Krause-Friedländer and Dejerine-Mayor state that a diminution occurs in the number of the cells, while Erlitzky, Drechfeld and Homén find that both processes occur together after amputation. The spinal, ganglion cell-bodies persist for some time after peripheral or distal fibres have degenerated (Friedländer and Krause, Homén, Vanlair, Marinesco).

Pelizzi[1] describes degenerative changes in the cord after amputation.

Having thus noted the types of change which have been described it will be well to briefly review the alterations which more recent investigators have found in individual cases.

Marinesco[2] examined three cases :—

(1) Male; amputation of middle of left thigh at 30. Death at 51 from dementia paralytica following an attack of apoplexy. Asymmetry of cord, left side smaller in lumbo-sacral region. The cells of the postero-lateral group in the anterior horn are only one-third of the number on the opposite side. Elsewhere a lesser degree of disappearance of cells on this side. Slight diminution in number of cells in left postero-lateral group in the cervical region. Little change in dorsal region.

(2) Male; amputation of middle of right upper arm at 40: death at 50. Atrophy of right side of cord in cervical region. Anterior and posterior nerve-roots atrophied (especially the latter). Diminution of the number of cells in the right lateral horn and also in the other cell-groups in the cervical region.

(3) Age at death and date of amputation [of left thigh] unknown. In the lumbar region a left-sided atrophy of the cord, not so marked as in the first case. No distinct change in the number of the anterior horn cells on the affected side. The cells

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[1] "Sur les modifications qui surviennent dans la moelle epinière des amputés" *Arch. ital. de Biologie.* Tome xviii., I.

[2] "Ueber Veränderung der Nerven und des Ruckenmarks nach Amputationen; ein Beitrag zur Nerventrophik." *Neurol. Centralbl.* B. xi., SS. 463-467; 505-508; 564-574, 1892.

of the posterior root-ganglia on the affected side appear intact. The neuroglia showed no hyperplasia.

Gregoriew[1] describes in detail the state of the spinal cord in five cases of amputation, two of upper limb, two of the thigh, and one of the leg. In four cases changes were found in the spinal cord; in one case of amputation of the thigh one year previous to death, the spinal cord appeared perfectly normal. The alteration observed consisted in atrophy of the elements of the cord on the side of amputation. The earliest parts affected were the posterior roots and columns. Later the motor portion of the cord was also affected and the intensity of the change depended upon the length of time elapsing since the amputation. The statement that the postero-lateral cell-group in the anterior horn was especially affected Gregoriew could not confirm; he found the ventral and lateral groups much more affected. In two cases degeneration of the posterior roots, slight in degree was observed, and in one case the peripheral nerves showed true degeneration and not mere atrophy.

Sibelius,[2] in a man 70 years of age dying from pneumonia, in whom five years before death the left lower extremity was amputated just above the knee-joint found that the anterior horn-cells in the lumbar enlargement of the cord were fewer in number on the side of amputation than on the opposite side. This diminution in number affected chiefly the postero-lateral group, but the other cell-groups were also affected. The changes in the anterior nerve-roots were unimportant, being considerably less striking than those in the posterior roots. Some of the nerve-cells in the ganglia on the posterior roots had disappeared and others had atrophied, on the side of the amputation in the segments corresponding to the limb removed. In the left posterior column in the same region there was a distinct atrophy with rarefaction of the nerve-bundles, and the course of this atrophy corresponded in the cord to that of the posterior roots derived from the affected ganglia. The reflex collaterals were fewer in number on the side of the amputation than on the opposite side. The cells in Clarke's column were also diminished in number on the side of the amputation. Sibelius regards the changes in the posterior columns, posterior roots and spinal ganglia as consisting of a degeneration of the spino-cutaneous neurons as a result of injury to their axis-cylinders in the peripheral nerves, in addition to which the trophic ganglia of these cells in the spinal cord also degenerate.

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[1] "Zur Kenntniss der Veränderungen des Rückenmarks beim Menschen nach Extremitäts-amputationen." *Zeitschr f. Heilk.* 1894, Bd. xv.

[2] "Till kännedomen om der efter amputationer uppkommande förändringrana i nervsystemet met special hänsyn till de spinokutana neuronerna." *Finska läkaresallsk handl.* 1897, xxxix., 10, S. 1879.

Elzholz[1] found in the stump of the limb after amputation several years previously [two cases were studied] that the large medullated fibres in the divided nerves became diminished in size as they descended in the stump, the diminution being due solely to thinning of the myelin sheaths, while at the same time a progressive diminution in number both of large and small medullated nerves took place. Non-medullated nerve-fibres made their appearance in increasing numbers as the extremities of the divided nerves were approached, these arising to all appearance out of the small medullated nerves.

The following is a summary of the changes, resulting from amputation of a limb, which the observers already quoted have noted :—

In the first place comes hemiatrophy of the spinal cord on the side of, and in the segments corresponding to, the lesion. A single exception is found in one of Gregoriew's cases, but as amputation occurred only one year prior to death it is probable that this change in the spinal cord had not had time to become manifest. The hemiatrophy affects both white and gray matter. In the white matter the nerve-fibres are atrophied and true sclerosis results.

Beyond this point there is difference among the observers quoted. Sometimes the posterior horn, sometimes the anterior horn, sometimes both together, have been found affected ; the nerve-cells have been found simply atrophied in some cases, while in other cases they have been found diminished in number on the side of amputation; in some cases the defect of cells has affected chiefly the antero-internal, in others the postero-lateral cell-groups of the anterior horn, while defect of Clarke's column has also been observed on the side of the injury. An exception to the usual diminution in number of the anterior horn-cells is found in Marinesco's third case.

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[1] "Zur Histolgie alter Nervenstumpfe in Amputirten Gliedern." *Jahrbücher f. Psych.* Bd. xix. Compare Marinesco, *loc. cit.*

The defect in the white matter has been noted to affect the posterior column corresponding to the side of amputation (Gregoriew, Sibelius and others), but the defect is not confined to this situation.

In animals amputation is followed by spinal cord changes similar to those occurring in man. These are more pronounced in developing than in adult animals.

The posterior roots at the level of the amputation are atrophied. True degeneration has also been described.

The ganglia on the posterior roots have, in different cases (1) appeared intact, or (2) presented both atrophy and defect of nerve-cells.

Degeneration has been met with in the peripheral nerves after amputation. Simple atrophy has usually been noted.

Little attention has been paid to the condition of the brain following upon amputation.

It is obvious from the foregoing that much further study of the changes in the spinal cord, posterior root ganglia, spinal nerve-roots and peripheral nerves in a larger series of cases is required before any very precise statement can be made of the extent and distribution in different cases of the structural alterations in the nervous system resulting from amputation, and the apparent discordance in the changes met with up to the present reconciled. Such observations are of very great importance in so far as they are likely to increase our knowledge, on the one hand, of the relation of the segments of the spinal cord to body areas and, on the other hand, of the connections and interdependence of the various neurones of the spinal axis.

It is very desirable that the condition of the brain after amputation should be also recorded. An examination limited to the spinal axis loses much of the value which it would otherwise have.

The case which forms the subject of the present investigation appears peculiarly fitted for the observation of some of the above points owing to the long period of time elapsing between amputation and death, and the excellent physical health and the very active life led by the patient up to within four years of death. On the other hand the condition of general brain atrophy accompanied by cortical softening in the left occipital lobe, which occurred towards the close of the patient's life, places a difficulty in the interpretation of some of the changes noted in the basal portions of the brain.

HISTORY OF CASE.

Patient was a male, 61 years of age at the time of death, whose occupation had been that of a house-to-house hawker, doing a large business with a horse and cart. When 19 years of age his right upper limb was amputated at the middle of the upper arm (fig. 1). Six years before his death he failed in business, became depressed, and after twice attempting suicide was admitted to the asylum. At the end of three months he was discharged. About a year after his discharge he suffered from influenza which left him irritable. Previous to this illness his general health had been good, and he had been for twenty years a total abstainer. Subsequently he deteriorated mentally and physically. His memory failed and he became demented. He was re-admitted to the asylum twenty-one months before death.

At the time of death there was a fairly extensive range of passive movement at the right shoulder-joint. The muscles of this shoulder were wasted to an extreme degree (fig. 1; note in particular the condition of the deltoid and pectoralis major), so that the head of the humerus and the rest of the bony skeleton of the shoulder were readily recognisable on inspection and palpation. The muscles attached to the left shoulder were of good development. There was some atrophy of the right trapezius, but the sterno-mastoids were of nearly equal development.

THE SPINAL CORD AND POSTERIOR ROOT GANGLIA.

The spinal cord upon removal showed some degree of asymmetry in the cervical region and upon close examination right-sided atrophy was observed. This condition was, however, far more readily noted after hardening when transverse sections

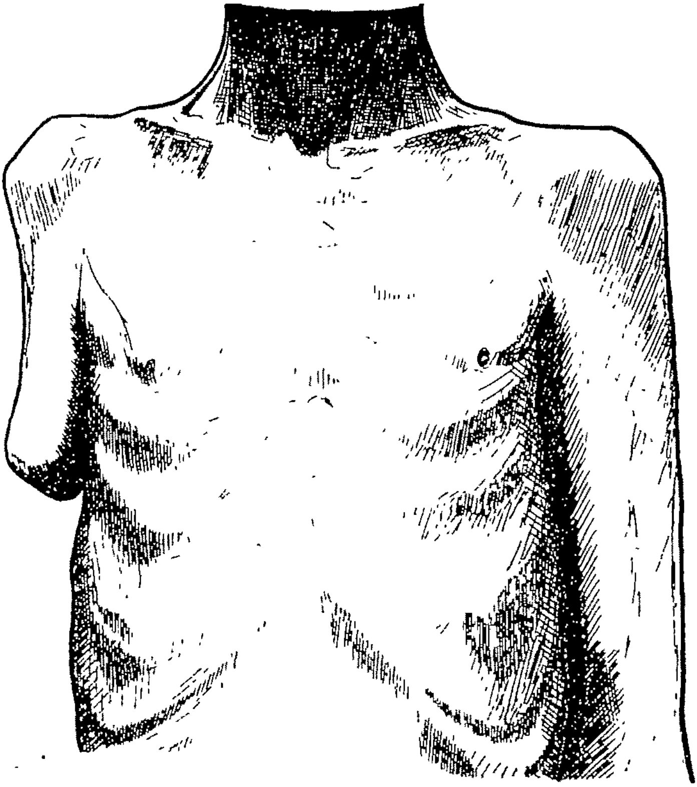

FIG. 1.

Sketch of the chest and the amputated right arm, together with the opposite upper limb, of the patient whose case is described in the text. The shoulder muscles on the right side are much atrophied, the contrast being very striking when comparison is made with the opposite shoulder. The right pectoralis major also is extremely wasted. The anterior edge of the trapezius is thin on the right side, but the sterno-mastoids are of equal size. The patient's general development is otherwise good.

were made.[1] Contrary to expectation there was no obtrusive difference in the aspect of the nerve-roots, posterior and anterior, on the two sides in the lower cervical and upper dorsal regions, but the anterior nerve-roots appeared on both sides somewhat small in this region.

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[1] The spinal cord after removal was hardened in alcohol. Subsequently each segment was sectioned transversely, the sections being stained by Nissl's method. In studying the condition of the nerve-cells as to size and number, numerous sections of each segment, made at varying levels of the segment, were examined and compared, it not being possible to draw reliable conclusions from the examination of single sections. Sections were also stained by v. Gieson's method, by carmine and by iron-hæmatoxylin.

The posterior root ganglia were examined by Nissl's and v. Gieson's methods.

Hemiatrophy.—The right-sided atrophy of the spinal cord (fig. 2) was strongly marked in the second to the sixth cervical segments, less marked in the next two cervical segments, slight in the first and second dorsal segments, and indistinct below the latter level. It affected both white and gray matter.

Posterior columns.—There was some wasting of the right posterior column in all the cervical and the first dorsal segments. Below the first dorsal segment the inequality disappeared. No marked sclerosis was, however, recognisable in the posterior columns. The position of the septum between the columns of Goll and Burdach was not sufficiently well defined to allow a positive statement to be made as to the existence of a localised atrophy in the right upper limb area of the former.

Antero-lateral columns. — The right antero-lateral column was smaller than the left. The wasting was noticeable in the cervical region and could be traced in the dorsal cord to the tenth segment, gradually diminishing in degree. The atrophy appeared to be largely in the situation of the crossed pyramidal tract (fig. 2) and was accompanied by an increase in the nuclei and staining capacity by Nissl's method of this area. Weigert's stain showed an increase of neuroglia fibrils. There was a similar sclerosis, much slighter in degree in the same situation on the left side, extending as low as the eighth dorsal segment The right antero-lateral column did not appear to be affected except in the situation of the crossed pyramidal tract. The portion of the right antero-lateral column lying between the anterior horn and the median septum is slightly narrower than its fellow in the cervical region. This difference disappears below the cervical region. The large size of the fibres of the direct cerebellar tracts permitted these columns to be recognised; no change was found on the right side in the amputation segments. The same held good of the antero-lateral ascending and descending cerebellar tracts.

Gray matter. Extent.—The gray matter of the right anterior horn was atrophied throughout the cervical, and in the first dorsal, segments. Below the first dorsal segment no well defined difference is recognisable. The right posterior horn was not apparently wasted. It was shorter and thicker than its fellow, but its superficial area in section was about equal to that of the opposite horn (fig. 2).

Anterior horn-cells.—The large multipolar cells in the anterior

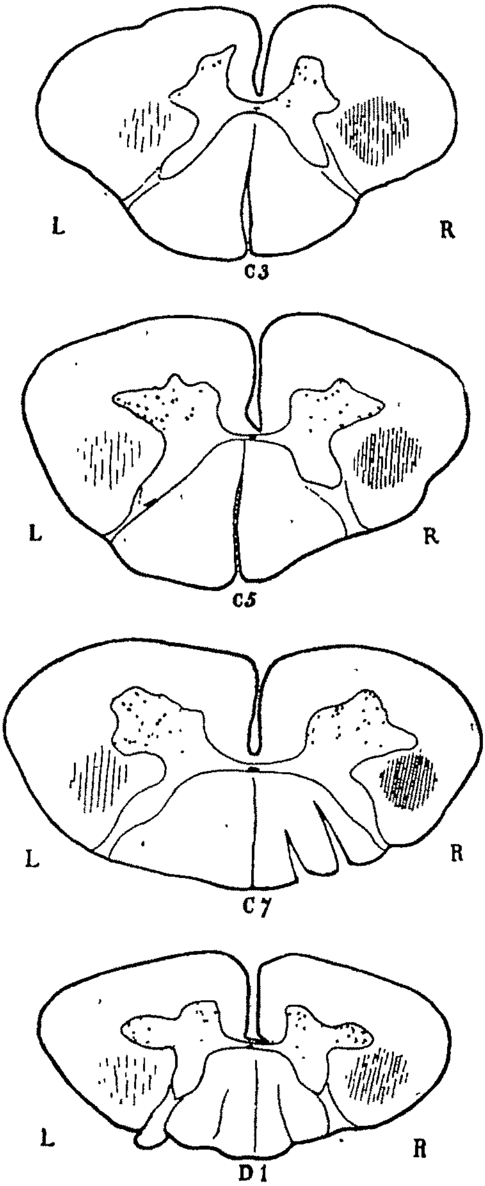

FIG. 2.

Outline sketches of transverse sections of the the third, fifth and seventh cervical, and the first dorsal, segments of the spinal cord, all drawn to the same scale, the large multipolar nerve-cells in the anterior cornua being also indicated in respect of size and number. There is hemiatrophy of the cord on the right side, both gray and white matter being defective. The diminution in size of the gray matter affects the anterior cornu, but not the posterior cornu to any marked extent. The large multipolar nerve-cells of the anterior cornu are also deficient on the right side, the defect in C 5 and C 7 being marked in the outer part of the anterior horn, and affecting both the size and number of the cells. Sclerosis of the right crossed pyramid tract is indicated by shading. A slighter degree of sclerosis is found in the left crossed pyramidal tract. R, right side; L, left side, in this and the succeeding figures; stained by Nissl's method. *Magnified five diameters.*

horn were diminished both in number and size on the right side. When a large number of sections from each segment are examined this diminution was found in the fifth; sixth, seventh and eighth cervical, and to a slight degree in the first dorsal, segments [1] This change was chiefly noticeable in the outer part of the anterior horn; the antero-internal cell-groups were more slightly if at all affected. Above the fifth cervical, and below the first dorsal segments, no constant difference on the two sides was recognisable; in the lumbar and sacral segments the cells on the two sides were equal. It is difficult to form a quantitative estimate of the difference on the two sides in the affected segments by counting the nerve-cells, owing to the difficulty of making a sharp separation between large and medium sized cells. I give, however, the following estimate of the relative number of multipolar anterior horn-cells on the two sides, obtained by counting as accurately as possible the number of these cells in forty successive sections from the eighth cervical segments, each 25 μ thick, those on the left side being taken at 100 for the sake of comparison. (The actual number counted on this side was 3,450.) The figures thus obtained, which must be regarded as only approximately correct, were: Right side, large 6, medium 77, together 83; left side, large 16, medium 84, together 100.

Posterior horns.—The cells in the right posterior horn showed no marked difference in number and size from those in the left posterior horn.

In consequence of the atrophy of the right anterior horn and the defect of white matter the spinal cord appears distorted on transverse section, and the anterior and posterior median septa tend to curve over to the right side in front and behind (fig. 2).

Sheath and blood-vessels.—At the necropsy the pia-arachnoid was found to be whitened and there was much cerebro-spinal fluid about the cord. On section the pia-arachnoid was seen to be thickened. The blood-vessels were thick-walled in the situation of the anterior septum. No change was noted elsewhere. The dura mater was unaltered in aspect.

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[1] In examining transverse sections note is taken only of such change in number and size of multipolar anterior horn cells as predominates in a series of sections of the same segment. Individual sections are not necessarily representative of the general condition of the segment in respect of its anterior horn cells.

Nerve-roots.—As already stated no marked one-sided defect in the size of the posterior or anterior nerve-roots was noted, though the latter seemed somewhat scanty on both sides at the cervico-dorsal junction. Nor were the root-fibres obviously of smaller diameter on the right side in the cervico-dorsal region.

Posterior root ganglia.—The posterior root ganglia could not be recognised to be smaller on the right side than on the left in the cervico-dorsal region. It is, however, difficult to observe a small alteration in their size accurately owing, on the one hand, to the adhesion of these ganglia to the dura matral sheaths requiring some dissection in order to expose the ganglia and, on the other hand, owing to the intermixture of nerve-cells and fibres. Microscopical examination also failed to reveal any constant or obtrusive difference on the two sides. The nerve-cells were of varying size consisting of large cells 78 μ to 50 μ in diameter, and smaller cells 35 μ to 25 μ in diameter, the former being relatively more and the latter relatively less numerous in the lower cervical and upper dorsal region than in the dorsal region below the upper two segments. The arrangement of chromophile material in the cell-bodies was unaltered on the right side, nor was the connective tissue increased. The appearance in transverse section of the mixed spinal nerves immediately beyond the ganglia was not markedly different on the right side from that on the left. No examination was made of the condition of the divided ends of the nerves in the stump.

THE BRAIN.

In considering the condition of the brain it will be convenient to note first of all the general appearance of the brain mantle and its coverings and then to study the changes present in the basal portion of the encephalon as exhibited in a series of transverse sections from below upwards.

Cerebrum.—The first point to note is the condition of senile atrophy which the brain (1,128 grammes) presented. The brain mantle was wasted generally, especially in the parietal and frontal regions, and corresponding to this wasting there was widening of the sulci and fluid in the meshes of the pia-arachnoid. The latter membrane was white and opaque at the vertex and to a less extent at the base where the cisterna magna and pontis contained much fluid. The ventricles were of large size, the left lateral ventricle being larger than its fellow (fig. 5). The ependyma was everywhere smooth. The choroid plexuses of the lateral ventricles were larger than normal.

There was old softening on the left side involving the apex of the cuneus and the adjoining part of the lingual convolution, extending to the ependyma of the lateral ventricle and reaching posteriorly to the optic radiation. With this exception focal lesions were absent from the cerebrum. The dura mater was adherent to the skull-cap and presented two calcified plaques, in the falx-cerebri, the larger being four centimetres long and the other slightly smaller; on the inner surface of the dura a thin rusty pellicle was present opposite the right temple and in the middle fossa. The basal vessels were generally thickened and stiff but presented very little nodular atheroma.

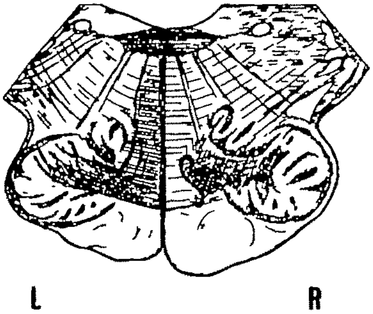

FIG. 3.

Transverse section of the medulla oblongata through the middle of the olivary bodies. On the left side the anterior pyramid is much smaller than on the right. The olivary bodies are of nearly equal size. The superior pyramidal decussation presents a slight inequality on the two sides, there being more decussating fibres on the left side of the median raphe than on the right. *Magnified three diameters.*

Medulla oblongata.—Turning now to the structures at the base of the brain, the medulla oblongata comes first for consideration (fig. 3). The left anterior pyramid was much wasted as compared with its fellow. This change was very striking. The left half of the section was smaller than the right, the difference being distinct though slight even in the dorsal region of the medulla. The olivary bodies were of nearly equal size. The superior pyramidal decussation was not symmetrical; more decussating fibres were seen on the left side of the median raphe than on the right. The hypoglossal and vagal nuclei were equal on the two sides, and the posterior column nuclei were also of nearly equal size.

Pons and cerebellum.—In the pons Varolii the same left-sided atrophy involving the pyramidal bundles, which here are of larger size than in the medulla oblongata, was observed (fig. 4). In the dorsal region of the pons there appeared to be little diminution of size, but the two halves of the section were not perfectly symmetrical; this want of symmetry did not appear to be wholly due to an obliquity in the plane of the section. The posterior longitudinal bundles were of equal size. There was a difference in the appearance of the fillet on the two sides apparently due to a difference in level (fig. 4). The cerebellum presented no change calling for note; the dentate nuclei were equal on the two sides.

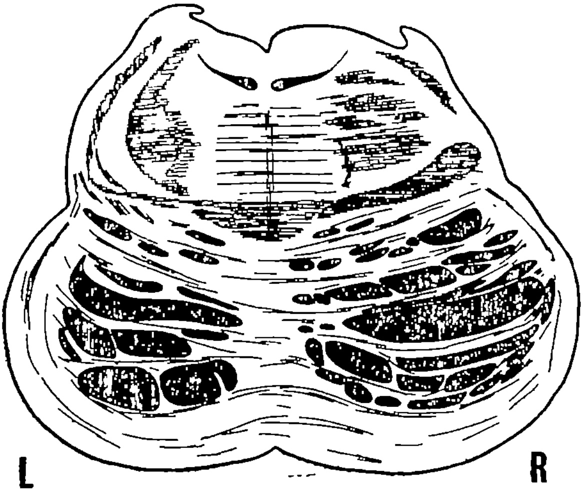

FIG. 4.

Transverse section of the upper part of the pons Varolii. The section is slightly smaller on the left side than on the right, owing to diminution in size of the pyramidal bundles. The dorsal portion of the pons does not exhibit a similar asymmetry. The fibres of the fillet are not cut quite at the same level on the two sides. *Magnified three diameters.*

Mesencephalon.—In the mesencephalon there was distinct atrophy of the pes pedunculi but the tegmentum was nearly equal in the two halves of the section.

Basal ganglia.—Both optic thalami were of small size (fig. 5). The left was however of much smaller size than the right, a difference which was much greater, when the thalami were viewed from behind, than would appear from fig. 5. This left-sided wasting affected especially the ventral portion of the thalamus and the pulvinar but the anterior and dorsal portions also suffered in a lesser degree. The corpora geniculata and optic tracts showed no marked one-sided wasting.

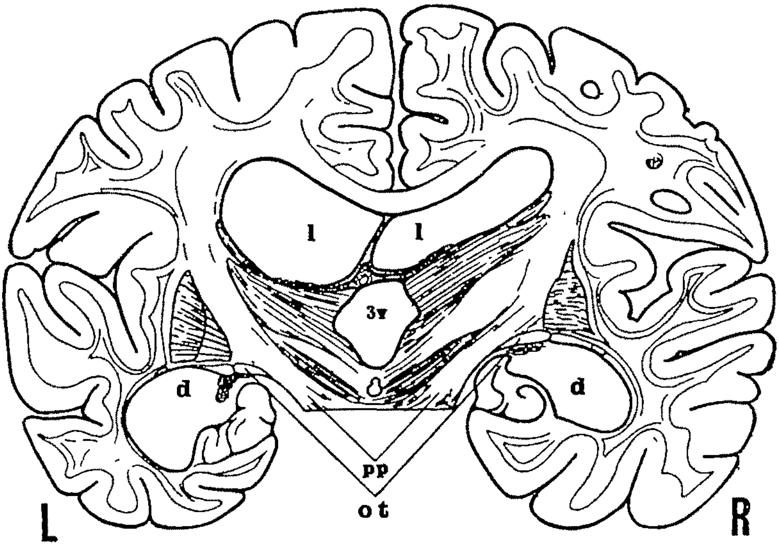

FIG. 5.

Frontal section of brain a short distance behind the corpora mammillaria. The sulci are widened and the lateral ventricles, *l l*, their descending horns, *d d*, and the third ventricle, 3 *v*, are dilated. The optic thalami lying on each side of the third ventricle are flattened. The left thalamus is smaller than its fellow, being wasted below in the subthalamic region. Below the thalami, there is seen on each side the locus niger, and externally and below this, the pes pedunculi, *p p*, is seen. More externally the lenticular nuclei are seen, and still more externally the islands of Reil; *o t*, optic tracts, external to which the inferior pedicle of the optic thalamus and the tail of the caudate nucleus are seen on each side forming the roof of the descending horns of the lateral ventricles. The opening seen below the third ventricle is the end of the posterior perforated space. *Slightly reduced in size.*

The corpora striata were of fair size and nearly equal, the left being slightly the smaller.

The internal capsule on the left side was not smaller than its fellow in the situation of the fibres going to the right upper limb.

There was a marked difference in the aspect of the optic radiation and inferior longitudinal bundles on the two sides, the left being rather less than half the thickness from side to side of the right.

The microscopical examination of the brain added little further information. In the medulla oblongata the right cuneate and gracile nuclei were not markedly different from the left. In the mesencephalon sclerosis could not be demonstrated in the left pes pedunculi. The left hand and forearm areas of the cortex, were not different from the corresponding areas of the right cerebral hemisphere.

The thoracic and abdominal viscera call for little remark. The kidneys show marked interstitial nephritis. There was pulmonary emphysema and congestion. The heart muscle was friable, and the mitral and aortic valves thickened. The body was somewhat emaciated, and the muscular condition was poor, but except in the region of the right shoulder, no unilateral wasting was found.

REVIEW.

The more important of the changes in the nervous system noted in the foregoing may now be recapitulated and at the same time some observations offered on their significance.

It may reasonably be suggested that the failure to discover any marked degree of atrophy of the posterior root ganglia corresponding to the portion of the right upper limb removed is to be explained by the difficulty of investigating the condition of these ganglia, the nerve-cells lying among abundant nerve-fibres and the ganglion being surrounded by stout connective tissue, so that a limited degree of atrophy may readily escape notice. The non-appearance of any marked diminution in size of the posterior roots corresponding to the four lower cervical and the first dorsal ganglia, comparison of which is much more readily made, cannot however be explained so easily. According to the few observations made upon this point, a disuse atrophy and disappearance of spino-cutaneous neurons is the usual consequence of amputation, but it does not appear that this event always occurs to a degree sufficiently marked to be with certainty recognisable. No such change, for example, was noted in Marinesco's third case;[1] and the observations of Elzholz[2] suggest that the posterior root ganglion cells, which as Dogiel[8] points out are not all spino-peripheral, are capable of preserving their integrity to a greater extent than might at first sight be expected. Having regard to these considerations, it seems logical to infer that in the present case atrophy of spino-peripheral neurons really existed, but in too limited a degree to be recognisable by the methods of investigation pursued. It is probable that the results of amputation in the human subject vary in degree in different cases. In the present instance the patient led a very active existence, and the tendency to degenerative changes may naturally be supposed to be less than in those cases in which an inactive existence followed amputation. It is to be noted also that no diminution, recognisable on inspection, in the size of the anterior nerve-roots in the cervico-dorsal region occurred on the right side, though the anterior horn cells exhibited a distinct diminution in size and number. Nor were the anterior nerve-roots diminished in diameter to any noticeable extent in the right cervico-dorsal region.

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[1] *Loc. cit.*

[2] *Loc. cit.*

Examination of the spinal cord is more readily made and is much more precise owing to the fact that here the two halves are placed side by side enclosed in a common connective tissue sheath. For this reason slighter degrees of atrophy are recognisable than is possible in the case of nerve-roots and posterior root ganglia. In the latter it is not possible to obtain with certainty sections of different ganglia at the same relative level, which are alone strictly comparable; and again atrophy of the ganglion-cells is recognisable only if considerable in degree and distribution.

The right hemiatrophy of the spinal cord in the case under discussion (fig. 2) affected the segments corresponding to the amputated limb, namely the fourth cervical to the first dorsal. It was not however confined to them, but involved also the remaining cervical segments, though not extending to the dorsal segments below the second. The hemiatrophy did not correspond in degree to the extent which the relation of the spinal segments to body areas would suggest. Thus

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[8] *Anat. Anzeiger* xii., p. 140, 1896. Also *Internat. Journ. of Anat. and Physiol.* xiv., p. 73, 1897; and xv., p. 345, 1898.

the eighth cervical and first dorsal segments were less affected than the segments immediately above. It seems clear that in passing from the lower to the upper segments of the cervico-dorsal region, the hemiatrophy becomes increasingly representative of the segments of lower level. This at once raises the question, which of the constituents of the spinal cord are chiefly involved in the hemiatrophy?

Taking first the gray matter, the atrophy here it will be noted corresponded to the amputation segments but also involved the cervical segments immediately above. The atrophy appeared to be confined to the anterior horn, not involving the posterior horn. The diminution in size and number of the anterior horn cells corresponded closely to the segments supplying the portion of limb removed and it did not extend beyond these limits (fig. 2).

As regards the white matter, the right posterior column showed atrophy in the cervical region, but no sclerosis was recognisable, and it was not possible to determine to what extent the upper limb fibres were involved. In the antero-lateral region sclerosis was recognisable in the situation of the right crossed pyramidal tract, and to a less extent in the left crossed pyramidal tract (fig. 2).

At this point it will be convenient to defer further consideration of the state of the white matter of the spinal cord until some features in the condition of the brain have been referred to.

The hemiatrophy present throughout the cervical region of the cord was seen again in the medulla oblongata (fig. 3), pons Varolii (fig. 4) and mesencephalon, and in these regions the side of hemiatrophy was changed from right to left, and was clearly due principally to atrophy of the left pyramidal tract. This was especially noticeable in the medulla oblongata where each pyramidal tract is concentrated into a single bundle. In a horizontal section of the internal capsules no difference was noted between the two sides; there was no recognisable narrowing in the situation of the upper limb fibres. In the hand and forearm areas of the cerebral cortex no marked difference was recognisable between the two hemispheres. Nevertheless, in the light of our present knowledge, the diminution in size of the left pyramidal tracts in the medulla oblongata, pons and mesencephalon must be attributed to atrophy of the corresponding cortico-spinal neurons, and perhaps of thalamo-spinal neurons also. And a similar explanation holds good in respect of the slighter sclerosis seen in the left crossed pyramidal tract of the spinal cord.

In the spinal cord again, as already mentioned, the right anterior horn in the upper limb segments was atrophied, some of the cells having disappeared or become reduced in size. It follows, therefore, that there was present in the case under consideration atrophy of two sets of motor neurons of different orders connected with the right upper limb: namely, cortico-spinal passing from the "motor" region of the cerebral cortex to the affected segments of the spinal cord; and spinal-peripheral connected with the muscle-innervation of the same region. The upper set of neurons corresponding to the opposite upper limb were also affected in a much slighter degree.

The question now arises, was this degeneration of neurons conditioned by the amputation? In the case of the lower set of neurons the distribution of the diminution in size and number of the right anterior horn cells in the segments of the cord appears to admit of no doubt that these changes were causally related to the amputation. Some doubt may, however, be felt as to whether the degeneration of the upper neurons was determined by the amputation, for the brain mantle had undergone senile atrophy. The marked wasting of the left anterior pyramid (fig. 3) is, however, strongly in favour of the view that the wasting of the left upper neurons was determined by the removal of the limb. Upon this point further observations in cases of old standing amputation will be of interest.

A reference to fig. 2 suggests that the hemiatrophy of the spinal cord is, as far as concerns the white matter of the antero-lateral column, largely due to atrophy in the situation of the right crossed pyramidal tract; at any rate such an explanation would account for the diminution in breadth of the right half of the spinal cord in the cervical region. Such an hypothesis could only be settled by the occurrence of a lesion which would define the crossed pyramidal tracts, such for example as a total transverse division of the spinal cord. In the absence of this, the exact effect of the sclerosis of the right pyramidal tract in respect of the altered form of the cord cannot be stated.

The condition of the direct pyramidal tracts is similarly difficult to determine in the absence of a lesion causing descending degeneration of these columns. There was little difference in the width of the anterior columns in the cervical region, but some asymmetry was present.

The direct cerebellar tracts which could be recognised by the large size of their fibres, were unaffected in the cervical region. The same is true of the antero-lateral ascending and descending cerebellar tracts.

The condition of the sensory path requires further consideration. The posterior column nuclei in the medulla oblongata showed no marked change in aspect. There was present slight left-sided atrophy in the medulla, distinct from and additional to the atrophy affecting the anterior pyramid (fig. 3). There was also a difference in the aspect of the superior pyramidal decussation on the two sides, the significance of which is not clear. In the pons Varolii and the mesencephalon there was no clear evidence of hemi-atrophy dorsal to the pyramidal tracts.

The left optic thalamus was much smaller than the right (fig. 5), the diminution in size affecting chiefly its ventral and posterior aspects but involving to a slight degree its anterior and upper portion also. This is presumably caused by the co-existing softening on the mesial aspect of the left occipital lobe, and does not appear to stand in any causal relation to the condition of the right upper extremity. The left optic radiation was conspicuously small. The co-existence of senile brain-atrophy places a difficulty in the way of determining the significance of changes in the basal regions of the brain. Further observations are required in old amputation cases in which this complication is absent.

Reviews.

Die Myasthenische Paralyse (Bulbärparalyse ohne anatomischen Befund). Von Prof. Dr. H. Oppenheim in Berlin. Mit 4 Abbildungen im Text und 3 Tafeln. Verlag von S. Karger. Berlin 1901.

This monograph will be welcomed by all who are interested in myasthenia gravis for its author has materially added to our knowledge of the disease and has by his previous writings largely contributed to its recognition as a distinct clinical entity. The work includes an abstract of all the cases hitherto reported.

The opening chapter is devoted to an interesting historical sketch, in which the part played by various observers in defining the disease as we now know it, is reviewed. After reference to the nomenclature, an abstract is given of the reported cases which appear to be undoubted examples of the disease; these are divided into three groups. Firstly, cases examined both during life and *post mortem* in which there could be no doubt as to the diagnosis (twelve); secondly, cases examined during life and *post mortem* which were in all probability examples of the disease (six); thirdly, cases in which although there was no *post mortem*, the clinical symptoms were sufficiently characteristic to make the diagnosis almost a matter of certainty (forty-one).

A short sketch of the chief clinical features and mode of onset of the disease is followed by a detailed description of the symptomatology based upon an analysis of the foregoing cases.

Among other points it is interesting to note the frequency with which the external ocular muscles are first affected (fifteen of fifty-one cases). In referring to the prominence of the eyeballs which is occasionally present in myasthenia gravis, the author remarks that this condition is not always associated with, and cannot therefore always be explained by, the presence of paralysis of the external ocular muscles; and he points out that other symptoms suggestive of exophthalmic goitre or, at all events, of a "forme fruste" of that disease are sometimes present.

The frequency with which death results from an attack of asphyxia is illustrated by a reference in tabular form to the eighteen fatal cases, in all of which death was due to this cause. Oppenheim is of opinion that true muscular atrophy should not be included in the symptomatology of myasthenia gravis; he thinks that in those cases in which a marked degree of muscular atrophy or qualitative disturbances of the electrical excitability were present there was probably some other disease or complication to account for their presence.

In the chapter on ætiology the author attaches importance to the neuropathic diathesis which, he believes, plays an essential part in the pathology. With regard to the influence of infective diseases as a causative factor, he remarks upon the probability that in some cases at least the myasthenia gravis was in existence prior to the onset of the infective disease, and its symptoms merely intensified by the acute intercurrent malady.

He points out that a thorough and detailed criticism of those cases in which pathological changes were met with in the nervous system fails to produce evidence of an organic lesion to account for the symptoms. The small hæmorrhages which have often been described in the central gray matter were, he thinks, probably produced during the death agony. He emphasises the occurrence in a few cases of congenital defects in the nervous system or other parts of the body as possibly throwing some light on the nature of the process. He suggests from a consideration of the pathological lesions which have been found in the other organs that here there exists a possible source for the production of a toxin. In common with the majority of writers Oppenheim regards the motor nerve nuclei or the whole of the corresponding motor neurons down to the muscles as the most probable seat of the lesion; while he classes the disease as a neurosis, dependent it may be to some extent on an associated congenital factor.

Before referring to the question of diagnosis an abstract of another series of cases is given. This fourth group includes the uncertain, doubtful, impure and mixed cases; these are considered separately either because of the existence of unusual symptoms or because the records are too imperfect to allow of certain classification.

The diagnosis is very fully treated of. It is pointed out that no single symptom is pathognomonic, but that the diagnosis must be based on the whole clinical picture and course of the disease.

Special attention is given to the differential diagnosis, and tables are inserted showing the chief points of distinction between myasthenia gravis, chronic progressive and acute bulbar palsy. After an exhaustive consideration of the very close resemblance which exists between some cases of polioencephalomyelitis and myasthenia gravis the author comes to the conclusion that they are two distinct and distinguishable diseases.

Referring to prognosis, the absence of any undoubted recorded instance of complete and permanent recovery is remarked upon, and the gravity of the prognosis in those cases where pharyngeal and respiratory paralyses exist is pointed out.

Under the heading of treatment, in referring to the danger which accompanies the passage of the stomach tube, the suggestion is made that if it is considered necessary to pass the tube the throat should be previously cocainised.

Reference is made to the harmful effect of faradism, to the possible beneficial effects of galvanism, and to the uselessness of massage.

Although it cannot be said that any drug has been shown definitely to have a favourable influence on the course of the disease, mention is made of the various medicinal remedies which have been hitherto employed.

A very complete bibliography, not only on myasthenia gravis, but on all the papers referred to in the monograph, completes this valuable treatise.

It is much to be wished that all German authors would adopt the simple, clear style which characterises all Professor Oppenheim's works and makes them so attractive to his foreign readers.

EDWIN BRAMWELL.

The Nervous System and its Constituent Neurones. By LEWELLYS F. BARKER, M.B.Tor., New York. (D. Appleton and Company, 1899, pp. 1084.)

THIS volume is stated in the title page to be designed for the use of practitioners of medicine and of students of medicine and psychology, and it is the outcome of a series of articles in the *New York Medical Journal.*

The book is divided into two parts. In the first 312 pages the newer conceptions of the histology of the central and peripheral nervous organs are reviewed, and in the second part the author states that "the attempt has been made to apply the neurone conception—that is the cell doctrine—as consistently as possible in the explanation and description of the complex architectonics of the nervous system."

We are glad that throughout the work the word *neurone* is used to mean a cell belonging to the nervous system with all its parts and is not restricted to any particular part of the nervous unit.

It would be quite impossible to give a detailed account of this volume, and only a brief mention of its contents can be attempted.

The first part consists of five sections. In the first section on the history of the development of the neurone concept, after describing the old theory of Gerlach of the universal network, the researches of His, Golgi, Forel, and Ramon y Cajal are given, and the theory of the last named author that the axis cylinders end freely. The vital staining of Ehrlich and the study of degenerations by the Marchi and Nissl methods are well illustrated.

The question as to whether the terminals of the axone of one cell fuse with the dendrites of another cell is discussed, and Held's observations of this important point is well explained, and with regard to Apáthy's views the author thinks that the verdict "not proven" should be given.

In Section II. the external morphology of the neurones is dealt with, and the ingenious method of determining by polarization the age of the myelinization of the nerve-fibres is referred to. This section includes the description of the cell body and its dendrites, of the axones, collaterals, side fibrils, and interneuronal substance. The author thinks favourably of a recent view propounded by Nissl that there are probably fibrillar structures within the protoplasm of nerve-cells and also existing as extra-cellular nerve-fibrils in the gray matter, to which he attributes the highest functions of the nervous system, but the whole matter seems very hypothetical at present.

Section III. describes the internal morphology of neurones, and thanks to Nissl's method of staining, a new language has sprung up to designate the different behaviour to this reagent of normal and abnormal constituents of neurones. This section almost more than any other shows the vast strides which the minute study of nerve-cells has made in recent years and to the average neurologist who has not made a special study of the matter a glossary would be necessary to understand such terms as a "somatochrome nerve-cell of the arkyostichochrome variety in the apyknomorphous condition" if Dr. Barker did not describe all these changes in most lucid terms and smooth over the difficulty of the words by giving the Greek derivations. In the last chapter of this section the author sums up in a comprehensive manner our present knowledge on the internal structure of the neurones.

Section IV. takes the histogenetic relation of neurones, describing the researches of His and others on the development of the spinal cord and medulla, of the peripheral nerves and the muscle masses. After thus taking the anatomy and histology of the neurone, we come, in Section V., to its physiology and pathology, and first to the metabolism and nutrition of the neurone, and then to its degeneration and regeneration. On the question of pathology the degenerations of Waller and Türck are described, but it is pointed out that, owing to improved methods of staining, the Wallerian degeneration theory will have to be modified, as it has been shown by various experiments that the nerve-cell is affected when its axone is divided, which is contrary to the doctrine of trophic centres formulated by Waller. The ablation experiments of van Gudden and Forel paved the way for this alteration in ideas, and Nissl is quoted as stating that changes in the cells of the facial nucleus can be detected as soon as twenty-four hours after the facial nerve has been divided.

Chapter XXI., in this section on the irritability of the neurones, is written in a highly philosophical spirit, and such subjects as the absence of complete repose of the neurones, as shown by the automatograph, and the dependence of muscular actions on the continuous passages of sensory impressions, as shown by Sherrington and Mott's experiments of dividing all the sensory nerves of a limb, are discussed, and also the "neurone threshold," or the degree of excitation of a neurone which just suffices to call forth a fruitful excitation, *i.e.*, a sensation or movement, in another neurone with which it is in contact. The author discusses the bearing of the neurone concept on Müller's doctrine of the specific energy of nerves, and gives it as his opinion—and one which seems most reasonable—that the constancy of the quality of the reaction, despite the variability of the external stimulus—as when the sensation of light is produced whenever the retina is stimulated—is due to the correlative differentiation of both the peripheral and the central organs, and not of one of these only.

In Chapter XXII., on the neurone as a unit of nervous function, the functions of the cell body and of its dendrites are discussed, and the original theory of Golgi, that the dendrites were purely for nutritional, and not for functional, purposes, is contested; and we think, with the author, that there can be no doubt that the observations of Ramon y Cajal disprove this theory. On the question of the conduction in the neurones in Chapter XXIII., the author considers that it would be very unwise at present to state positively that nerve impulses may not pass in both directions in all neurones. The last two chapters of this section are on most interesting subjects, the relation of trophic to nervous functions in the neurone—where examination of nerve-cells in tired and normal animals shows an intimate relationship between states of nutrition of the cell and functional activity—and the histological alterations in neurones, due to action of poisons, compared with traumatism.

The rest of the work is taken up with "the grouping and chaining together of neurones in a complex nervous system like that of man and mammals." This is a minute description of the different tracts of the nervous system and their end stations, which have been found out by the methods of development or degeneration, and staining by various ways. The description begins with neurones connecting the sense organs of the body with the central nervous system; then the neurones within the central nervous system connecting the end stations of the axones of the peripheral centripetal neurones with other portions of the central nervous system, and goes on to the neurones directly connecting the central nervous system with the voluntary muscles of the body (lower motor neurones), and those which enter into conduction relation with the lower motor neurones and throw the latter under the influence of other centres (intermediary and upper motor neurones); and, lastly projection, commissural, and association neurones.

The author has not touched upon the structure of the sympathetic system, and as the work is devoted to histology and anatomical arrangement, no mention is made of the chemical side of neurology, with the exception of the tigroid matter in Chapter XII.

We have nothing but the highest praise for this work, and we congratulate Dr. Barker on the valuable contribution which he has made to neurology in America.

The book is not a mere compilation of facts, but on all doubtful points we have the advantage of Dr. Barker's opinion, which much enhances the value of the work. The references are exceedingly numerous, and no pains have been spared to give credit to the workers of all nations, and, consequently, the labour involved must have been very considerable.

The illustrations, of which there are no less than 676, with two coloured plates, play an important part; the reproductions of the originals have been most faithfully carried out in the case of those taken from other sources, and of the drawings made especially we would note the series of sections made through the new-born medulla and stained with Weigert-Pal.

The great strides which have been made in the minute anatomy, owing to the improved methods of staining and the effect of this on the theory of the nervous system, are well shown by comparing this work with text-books of, say, ten years ago. There is probably no department of histology where the revolution has been so complete, and this difficult subject has been here treated in a most lucid and masterly fashion, and the result is a text-book, which workers in neurology on both sides of the Atlantic will find of the greatest help in ascertaining the latest views on the minute structure of the most complicated parts of the nervous system.

C. E. Beevor.

Semeiologie du Système Nerveux. Par J. Dejerine, *professeur agrégé à la Faculté de Médecine de Paris, Médecin de la Salpêtrière.* (Paris: Masson et Cie. 18 francs).

This remarkable work forms the greater part of the fifth volume of the "Traité de Pathologie Générale," published by Professor Bouchard. It extends to eight hundred and twenty pages of text and contains three hundred and six illustrations. Its plan is a complete departure from the usual method of treating the diseases of the nervous system, for the author has limited himself entirely to the consideration and illustration of the symptomatology of the disorders of its various great functions.

The subject has been considered under the following heads: affections of intelligence; of speech; of the motor functions; of the sensory functions; of the reflexes; of the viscera, in so far as they are of nervous origin; of the trophic functions; and there is a special concluding chapter on the nervous affections of vision and the visual apparatus.

Under the disorders of intelligence are considered coma, sleep, insomnia, trance, somnambulism, ambulatory automatism, hypnotism and suggestion. Those affections which constitute insanity are beyond the scope of the work and are therefore not included.

The affections of speech are discussed under the heads of aphasia, dysarthria, mutism and stammering. The first of these, aphasia, is treated with great detail as to the differential diagnosis and localisation of its various forms and its progress. The author reiterates his views that there is no specialised graphic centre similar to the speech centre in Broca's convolution.

Nearly four hundred pages are devoted to the disorders of the motor functions, viz., paralyses, atrophy, ataxy, vertigo, contracture, convulsion, tremor, &c. There are special chapters on the anatomical localisation of the lesions which produce paralysis and muscular atrophies, and on the symptomatology of the hand, foot, face, attitude and gait, as well as on the mechanical and electrical reactions of nerves and muscles. This section of the work is a veritable storehouse of information on every disease and modification of the motor functions, and the illustrations, which are both numerous and apposite, are reproduced with a clearness that one has seldom, if ever, seen equalled.

The disorders of sensibility have been considered under the sections of superficial, deep and visceral sensibility, subjective disorders and localisation. Special attention is drawn to the investigation of the osseous sensibility and its relation to the deep reflexes—a relation which has been studied in Professor Dejerine's clinique by one of his pupils, but which does not yet appear to have attracted the attention which it deserves. A special chapter is devoted to the localisation of the lesions in the peripheral nerves, roots, cord and brain. The superficial and deep reflexes are discussed almost entirely from the clinical standpoint, as they are found in general diseases as well as in the organic and functional diseases of the nervous system. The relation of these reflexes to flaccid and rigid paralysis is more fully considered in the section on contracture (p. 265).

The visceral disorders (which are for the most part of a functional nature) and the trophic affections also receive adequate examination, as do the affections of the motor, sensory and central apparatus of vision.

The whole subject, thus hastily sketched, has been treated in a most masterly manner, with all the thoroughness, sense of proportion, and wealth of detail which Professor Dejerine's previous published work has led us to expect. The result is the production of a work which is at once unique and invaluable, not only to the student and teacher of nervous disease, but also to the busy general practitioner, who will find here, ready to hand, a series of clinical pictures of nervous diseases, such as are only to be found together in large special hospitals, and he will also find, in a singularly clear and accessible form, most of the data for differential diagnosis. It is the most complete clinical encyclopædia of nervous diseases that has yet appeared in any language.

Of the illustrations which accompany the work it is difficult to speak in terms of sufficiently high praise. They are reproduced in a uniformly high standard of artistic excellence.

We trust that the work may be made accessible to a wider circle of readers in an English translation.

ALEXANDER BRUCE.

A Topographical Atlas of the Spinal Cord. By ALEXANDER BRUCE, M.D. (Williams & Norgate, London and Edinburgh, 1901.)

IN the Atlas before us the author's aim has been to provide representations of sections which may be taken as typical of each of the segments of the spinal cord, with a view to determine the relative arrangement of the gray and white matter, especially the form of anterior horn characteristic of each segment; the relation and distribution of the motor nerve-cells; and any other features which may serve for the identification of the several segments. In this way a series of normal standards have been provided which will prove not only highly useful for the identification of sections submitted for examination, but will also serve as a basis of comparison with pathological conditions, and as an aid to the study of localisation of the motor functions of the spinal cord.

The determination of the exact locality of a given section of the cord may be made from the type of outline of the anterior horn, which is found to be constant for each segment or for corresponding parts of segments in different cords. It is only in the dorsal region, where the transition from segment to segment is less abrupt, that differentiation may become a matter of difficulty. The mesial column of cells persisting throughout the entire length of the cord, affords evidence that it supplies the muscles of the trunk; the lateral cell-columns being more closely related to the limb muscles. In the limb enlargements, the gray matter is increased by the accession of a promontory on its lateral aspect. It would appear also as if the groups of cells supplying the flexors of the limb were situated in the posterior part of the anterior horn, while those supplying the extensors lay in front. It is probable also that there is some relation between the size of the motor cell and that of the muscular fibre which it innervates. This would explain the small size of the cells in the first dorsal segment, as compared with the rest of the cervical region.

The Atlas consists of thirty-two plates, one plate being given over to the delineation of a typical section from each of the segments of the cord. In the upper part of the plate there is a photograph of the arrangement of the cells of the anterior cornua, and in the lower a transverse section of the cord, giving the outline of the gray matter. A short description of the plate is found on the opposing page. The figures are photogravure reproductions of the original sections.

This Atlas is another instance of the careful and elaborate work which we associate with the name of the author.

W. A. Turner.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

A PATHOLOGICAL MEETING was held at 11, Chandos Street, on Thursday, May 9, at 8.30 p.m., when the following communications were made:—

(I.) Dr. JOHN TURNER.—*A method of staining nervous tissues in methylene blue, whereby one is able to demonstrate:*

(1) The beaded network around the pyramidal nerve-cells and their processes;

(2) The "thorns" on the branches of Purkinje cells;

(3) And the basket-work around these latter.

The method appears to exert a selective influence in its action, staining some cells very darkly, others very lightly. The pyramidal and the Purkinje-cell systems stain light. The basket cells and small cortical cells of the cerebellum and certain small, square, pentagonal or pyriform cells of the cortex cerebri stain densely.

The staining appears to be permanent.

The drawbacks to it are that only parts of the tissue take on the stain. Neither the first layer of the cerebrum or the granular layer of the cerebellum stain.

Apparently it can only be got when the light and temperature are favourable, so that whilst in the spring, summer and early autumn successes are the rule, in the winter months and during cold dull weather they are the exception

(II.) Dr. MICHELL CLARKE.

(1) *Case of pachymeningitis of the spinal cord.*—From an agricultural labourer, aged 26. He had had rheumatic fever twice, had been much exposed to cold and damp, but denied syphilis. Duration of illness about eighteen months. Onset with weakness in legs, then shooting pains in arms, legs, and

trunk, tingling and numbness in legs, and some pain in back, with girdle pain about level of umbilicus. Gradual increase of paralysis in legs, with increase of deep reflexes, wasting of muscles, anæsthesia over certain areas, with patches of hyperæsthesia, and retention of urine.

Two months before death paralysis extended to upper extremities, with tingling and numbness, and shooting pains in them, followed by wasting of muscles. Later paresis of diaphragm.

For the last month almost total paralysis of limbs, with contraction and wasting. A marked clinical feature of the case was the extraordinary number of bedsores, which in spite of every precaution, formed on every bony point on which pressure occurred. There was disease of the mitral valve.

Post mortem.—The meninges covering the brain were normal. The ventricles, especially the lateral ventricles, were distended. The brain was rather small, but healthy in all parts.

Spinal cord.—Dura mater appeared normal externally, but on making transverse sections the cord was found to be enclosed from end to end in a dense fibrous envelope, measuring where thickest one-eighth of an inch in diameter. This envelope is formed partly of thickened dura mater, but chiefly of moderately dense newly formed connective tissue lying between the dura and the pia, which is also thickened. This new formation was thickest on the posterior aspect of the cord, and in the dorsal, upper lumbar and lower cervical regions, gradually ceasing at the upper cervical and sacral regions. The anterior and posterior nerve roots, and in parts most affected the posterior ganglia, were compressed and more or less destroyed; the outer margin of the cord to the extent of about one-eighth of an inch was destitute of nerve-fibres. The cord itself was compressed, its framework increased in density, and there was extensive atrophy of the cellular elements of the gray matter.

(2) *Changes in posterior columns of spinal cord in a case of pernicious anæmia.*—Sections show early stage of degeneration in posterior columns in cervical region (lower part chiefly) and cervico-dorsal region, not extending below second dorsal roots.

With the exception of slight tingling in hands and feet no symptoms of disease of the cord were present during life.

(3) *Tumour of the right optic thalamus*, involving the subthalamic region, and pressing on the right internal capsule, extending backwards it invaded the left subthalamic region, right

crusta, gray matter of origin of third nerves, and compressed the aqueduct of Sylvius. The corpora quadrigemina were flattened and displaced upwards.—Duration of illness two and a half years. Absence throughout of optic neuritis, vomiting and severe headache.

Symptoms in order of onset.—Diplopia, paresis left third nerve; paresis, burning pains, defective muscular sense and tactile sensation left arm and hand; horizontal nystagmus; paralysis left third nerve. *Very marked tremor, during movement only, of left upper extremity, exactly resembling tremor of disseminate sclerosis.* Speech deliberate. Want of variation in expression in features. Ptosis on both sides. Left hemianæsthesia. Left hemiplegia with exaggeration of deep reflexes.

Paralysis of both third and fourth nerves. Immobile dilated pupils. Finally paralysis on both sides of body, contracture, incontinence, drowsiness and coma.

(III.) Dr. Wakelin Barratt.—*Changes in the nervous system in a case of old-standing amputation.*

Clinical account.—Patient's upper limb was amputated at the middle of the upper arm when he was 19 years of age. He was in good health and led a very active life for thirty-six years afterwards. He then gradually became demented and died at the age of 61.

Post-mortem appearances.—There is hemiatrophy of the spinal cord on the side of the amputation in the whole length of the cervical region. In the medulla and pons there is atrophy of the pyramidal fibres of the opposite side. The hemiatrophy of the spinal cord affects both gray and white matter, the former showing diminution in number and size of the anterior horn cells.

There is little difference in the aspect of the posterior root ganglia on the two sides, nor is any striking change noticeable in the appearance of the nerve-roots, posterior and anterior, on the side of the amputation.

The brain is wasted and the pia-arachnoid at the vertex is opaque and thickened.

(IV.) Dr. Frederick Batten.—*Specimens from three cases of infantile paralysis.*

(1) Thirteen days after onset of disease.
(2) Three months after onset of disease.
(3) Two years after onset of disease.

An Extraordinary Meeting was held in the Medical Department of the Owens College, Manchester, on the morning of June 22, 1901, when the following specimens were shown:—

Dr. A. Bruce.—(1) Photogravures of each segment of the normal spinal cord. (2) Photographs and sections of: (*a*) Hæmorrhage into spinal cord. (*b*) Cavities in cord above a spina bifida. (*c*) Syringo-myelia. (*d*) Progressive muscular atrophy and amyotrophic lateral sclerosis. (*e*) Glioma of pons. (*f*) Syphilitic meningo-myelitis. (3) Photographs from a case of complete paralysis of the brachial plexus.

Dr. Judson Bury.—(1) Porencephalus. (2) Hydrocephalus. (3) Acromegaly.

Professor Sheridan Delépine.—(1) Actinomycotic abscess of brain. (2) Experimental arsenical neuritis. (3) Other lesions of the central nervous system.

Professor J. Dreschfeld.—(1) Spinal cord in ataxia paraplegia. (2) Spinal cord in disseminated myelitis. (3) Casts and photographs.

Mr. W. Milligan.—(1) A portable surgical motor.

Dr. F. Craven Moore.—(1) Multiple gummata of brain and spinal cord. (2) Hæmorrhagic meningo-encephalitis (influenza). (Dr. Dreschfeld's case.) (3) Metastatic calcification of vastus internus tendon with Charcot's arthropathy. (Dr. Dreschfeld's case.) (4) Specimens from poisoning by arsenical beer. (5) Cerebral tumours.

Dr. F. W. Mott.—(1) Effects of bitulin, abrin, absinthe, and picrotoxin upon nerve-cells.

Drs. Orr and Rows.—(1) Types of nerve-cells in the spinal ganglia of rabbits. (2) Nerve-cells in the spinal ganglia of man and different forms of chromatolysis in general paralysis of the insane. (3) Spinal ganglion cells in acute mania, showing vacuolation.

Dr. E. S. Reynolds.—Photographs of neurological cases, including a series illustrative of the recent epidemic of arsenical poisoning.

Professor C. S. Sherrington and Dr. E. E. Laslett.—Microscopical sections (*infra*).

Professor W. STIRLING.—(1) Exhibits to illustrate some of the phenomena of stereoscopic vision. (2) An original experiment on binocular colour vision. (3) An experiment to show how the actual size of the pupil is affected by the cornea. (4) View of the interior of the frog's heart to show the arrangement of its muscular fibres. (5) Enucleation of the eyeball and removal of the floor of the orbital cavity, showing position of soft palate in phonation. (6) Simple method of examining the retina in a living rabbit. (7) A classical experiment concerning the discovery of the capillaries (Malpighi, de pulmonibus) and a modern one. (8) Some optical illusions. (9) Some experiments in physiological optics. (10) Lantern slides of some distinguished physiologists. (11) Original picture of the Kircher's "Experimentum Mirabile." (12) Some anaglyphs. (13) The structure of some electrical organs. (14) Complete injection of the blood-vessels of the eyeball of a small animal.

Dr. E. F. TREVELYAN.—(1) Various specimens showing the value and permanency of Laskowsky's method. (2) Specimens preserved in Kaiserling's fluid. (3) Tuberculous tumour of the brain, meninges and skull. (4) Intact pia-arachnoid from a fœtus.

Prof. A. H. YOUNG.—Stereoscopic photographs of the brain.

Dr. W. B. WARRINGTON.—(1) Sections of unilateral atrophy. (2) Photographs of Tooth's peroneal atrophy.

Dr. R. T. WILLIAMSON.—(1) Two cerebellar tumours with photographs. (2) Microscopical specimens of:—(*a*) Progressive muscular atrophy. (*b*) Early tabes. (*c*) Acute anterior poliomyelitis of the adult. (*d*) Myelitis in the distribution of the anterior and of the posterior spinal arteries. (*e*) Peripheral nerves in multiple neuritis. (*f*) Spinal syphilis—thrombosis, hæmorrhage and myelitis. (*g*) Retro-bulbar optic neuritis associated with cancer of stomach.

Clinical cases were shown by :—

Dr. ASHBY.

Dr. JUDSON S. BURY.—(1) Syringomyelia with hemianæsthesia. (2) Sensory aphasia (two cases). (3) Ophthalmoplegia. (4) Hemiplegia, hemianæsthesia and impairment of special senses on one side. (5) Cases of muscular atrophy. (6) Hæmorrhage (?) into the spinal cord.

Dr. DRESCHFELD.—(1) Thomsen's disease (two cases). (2) A family (five cases) showing various stages of Erb's muscular dystrophy.

Dr. HARING.

Dr. HARRIS.—Unusual defect in the skull (? cephalhydrocele) with both hysterical and epileptic attacks.

Dr. DIXON MANN.

Dr. E. S. REYNOLDS.—(1) Locomotor ataxia with multiple joint lesions. (2) Unilateral lesion of the spinal cord (two cases). (3) Ataxia paraplegia (disseminated sclerosis?). (4) Chronic anterior polio-myelitis.

Dr. R. T. WILLIAMSON.—(1) Unilateral tremor most marked in leg (? paralysis agitans). (2) Very early tabes with knee-jerks present (two cases). (3) Disseminated sclerosis following influenza.

Dr. WILLIAMSON and Dr. P. TYTLER.—Hydatid of spine (extra-dural) with compression myelitis. Operation with gradual improvement for twelve months.

Prof. C. S. SHERRINGTON and Dr. E. E. LASLETT gave a short "note on the third, fourth and sixth cranial nerves."

Drs. ALEXANDER BRUCE, MOTT and ORR gave lantern demonstrations of specimens shown in the Histological Laboratory (*supra*).

Prof. WM. STIRLING showed (1) Some classical experiments bearing on neuro-physiology, and one new experiment. (2) Malapterurus electricus (living) and its mode of feeding.

BRAIN.

PART III., 1901.

Original Articles and Clinical Cases.

CERTAIN MENTAL CHANGES THAT ACCOMPANY VISCERAL DISEASE.

(The Goulstonian Lectures for 1901.[1])

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INTRODUCTION.

CHAPTER I.—CHANGES THAT MAY ACCOMPANY VISCERAL DISEASE.
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INTRODUCTION.

WE are all conscious that our mental well-being is dependent on the right activity of our internal organs, and daily experience shows that grave mental disturbances may arise in connection with visceral disease.

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[1] Delivered before the Royal College of Physicians, June 25, 27, and July 2, 1901.

Such mental changes may be produced in many different ways.

(1) A disturbed activity of the heart may alter the circulation to the brain or may change the character of the blood with which it is supplied. The delirium that accompanies a failing heart or profound vascular degeneration may be instanced as an example of a mental change dependent directly on altered vascular conditions.

(2) Disease may attack some organ which in health produces a substance necessary for the normal metabolic activity of the tissues. Thus, destruction of the thyroid gland is followed by the characteristic hebetude of myxœdema owing to the absence from the body of some constituent manufactured by that gland.

(3) Disease may attack some excreting organ and thereby lead to the retention of toxic agents in the blood.

Such toxic substances are probably responsible for the morbid mental states which frequently accompany disease of the kidney and liver.

(4) Visceral disease may so lower the resistance of the nervous system that a poison such as alcohol, already circulating in the blood, can produce mental changes differing perhaps both in quality and intensity from those which would have been produced by the same poison under other conditions.

(5) Lastly, disease of the viscera may so lower the patient's resistance that some inherent mental tendency may become manifest as active mental disease.

It might be thought that all changes in mental states directly traceable to the presence of some visceral disease could be ascribed to one or more of the above causes.

But now that disease of internal organs has been shown to be accompanied by pain radiating around the surface of the body and by tenderness of its superficial coverings it becomes necessary to examine in how far the intrusion of such stimuli upon the nervous system are accompanied by changes in consciousness. It is my intention therefore to bring before you in these lectures a group of changes in consciousness mainly associated with the reflected pains of visceral disease. These mental changes are recognised by the patients themselves, and in some cases are associated by them with the first appearance or progress of their disease. Yet in spite of the frequency with which these symptoms make their appearance, their existence is unknown to the physician and to the experimental psychologist.

But before passing on to the results of this investigation, let me first say a few words on the method by which they have been obtained.

The work was begun in 1893, when, as House Physician at the Victoria Park Hospital for Diseases of the Chest, I had the opportunity of seeing many cases of disease of the heart and lungs under the most favourable conditions for accurate observation. Owing to the kindness of the Staff of this Hospital, and the liberality of the Committee, my period of office was extended, and I was permitted to continue my investigations even after I ceased to be their officer.

For the last five years the observations made at the Victoria Park Hospital have been amplified and confirmed at the London Hospital, both from the study of patients under my own care and those under the care of my colleagues.

The patients I have studied for this research belonged to the usual hospital class, and more particularly comprised such members of this class as suffered from pulmonary, cardiac, or abdominal disease. I have included no patient who had been insane or was suffering from any form of nervous disease. I have excluded all those with an hereditary taint of insanity, and all Hebrews, owing to the recognised tendency of this race to functional neuroses.

In every case the notes were taken by myself, and it is useless for any one to attempt to either controvert or confirm the results I am about to bring forward if he delegate that work to another. The usual history taken by a ward clerk or registrar is useless for this purpose; for the patient is extremely sensitive on all matters that concern the sanity of his mental processes, and it is only after his confidence has been gained that the full extent of the mental changes can be estimated that underlie an apparently normal behaviour.

Above all things it is necessary to avoid allowing the patient to think you believe him to be mad. I therefore never take the family history of neuroses and insanity until the whole examination is finished.

It is absurd to suppose that he will confess to the existence of mental changes before a crowd of students such as accompanies a physician in his usual visit to the wards. And in most cases it is not advisable that the physician when investigating a patient from this aspect, should be accompanied even by a sister or nurse; for men are peculiarly adverse to exposing their emotional weakness before a woman, and women do not trust another woman not to tell "how silly they have been." But both male and female patients have no reticence before a capable house physician, recognising that the treatment of their disease depends on his complete knowledge of all its phenomena, however remote. Thus, in order to control my observations, I have of late frequently examined into the mental state of these patients in the presence of my house physicians.

The publicity of a large hospital ward is a bar to confidence, and I am therefore in the habit of placing screens round the patient's bed during such examination. For the same reason darkness is a great help to confidence, and many of my notes have been taken between the hours of seven and nine in the evening.

I am in the habit of begining with the history of the present illness and previous ailments. I then ask if the patient has been low spirited, and estimate in conversation with him the presence or absence of causes that may have led to such depression. Thence I pass to sleep or dreams, and so gradually to enquiries for hallucinations, loss of memory or attention, and delusions of suspicion. If the patient shows any disinclination to answer or fear as to what his answers may lead me to think of his mental capacity I question him with regard to his life or perhaps pass on to a physical examination. However, as a rule, if precautions are taken to ensure privacy, patients show no reluctance to reveal symptoms that many of them definitely associate with the oncoming of their disease, provided the interrogator be the physician under whose care they may be during their stay in a hospital. To many of them it is a relief to talk freely of phenomena that have puzzled or frightened them; phenomena of which the description has exposed them to universal ridicule or to the accusation of untruthfulness.

No one can have worked in the wards of a hospital without recognising the apparently inexplicable changes in character that from time to time occur amongst the patients. On passing from bed to bed in the usual round you may have been astonished to notice the change in some patient suffering from disease of the lungs or of the heart. The day before he may have been sitting up in bed quiet, orderly, answering each question well. Now he is lying with face averted, obviously unwilling to be disturbed. His answers are monosyllabic, and if examination be persisted in he is seen to be keeping back his tears with difficulty. The sister of the ward says that "Number so-and-so has been a little hysterical lately," and so the matter drops. On your next visit you may find that this patient has discharged himself without lodging any complaint or giving any reason.

But if you go back to this patient later in the day you will frequently find that he has resumed his usual condition, and if you are unattended by students or nurses he will willingly confide to you the curious symptoms by which he was attacked. This he will the more readily do as he feels he owes you an explanation for his otherwise inexplicable conduct earlier in the day.

He will tell you that he suddenly became low-spirited, and was sure something was going to happen to him. The nurse and sister of the ward are "against him" and "want to get rid of him." His sleep has been disturbed by horrible dreams, and he may tell you that one night he woke to see a corpse standing by his bed. He has not been able to take his dinner because everything stank. Lately he has frequently imagined filthy smells that made him feel sick. No one else can ever smell them. Moreover, his memory has become very bad lately, and he reads the same thing in the newspaper over and over again.

Now, I hope to be able to show you that in most cases the reflected pain of visceral disease is the main factor in the production of these mental changes.

CHAPTER I.—CHANGES THAT MAY ACCOMPANY VISCERAL DISEASE.

§ 1.—HALLUCINATIONS.

UNDER certain conditions sane persons suffering from visceral disease are liable to develop hallucinations of sight, of hearing, or of smell. Hallucinations of taste also probably occur, but I shall neglect the possible occurrence of this form of hallucination. For, owing to the condition of the tongue, it is usually impossible to be certain that the sensation of which the patient complains has no objective basis.

The precautions taken to insure accuracy have been unusually stringent. All observations on hallucinations are, in my records, divided into three classes according to the conditions under which each observation was made.

In the first class stand those cases where the patient was under observation in the hospital at the time the hallucination occurred; where it was demonstrably certain that the sensation had no objective basis and where the physical state of the patient, more particularly the condition of the sense organ implicated, and the sensibility of the skin were recorded both before and after the supposed hallucination.

In the second class I have placed such hallucinations as occurred during the patient's stay in hospital under continuous observation by myself, but where my attention was not called to the occurrence of an hallucination until more than twenty-four hours after the event.

The third class contains cases where the patient describes the occurrence of what appear to have been hallucinations before his entry to the hospital. It is obvious that the value of this group will vary according to the form assumed by the hallucination. Thus, if a patient states that when at home he suffered from the occurrence of filthy smells that no one else could appreciate, his statement is of little value for this research. But if, on the other hand, he woke one night to see a white figure standing by his bed which his wife was unable to see, there is a strong probability that he was subject to an hallucination of vision.

Thus, when discussing hallucinations of vision I shall make use of all three classes. When dealing with hallucinations of hearing I shall use only those that occurred during the patient's stay in hospital, but shall touch for comparison on certain occurrences that were demonstrated by the patient and his friends to have had no objective basis, or were opposed to natural probability (*e.g.*, the ringing of a bell in a house that has no bells). With regard to hallucinations of smell, I shall only deal with those where I was able to prove by examination at the time of their occurrence that there was no external cause for the smell of which the patient complained, nor any condition of the nose, mouth, or tongue that could in any way be held responsible for its production.

It might be justly urged that such canons of criticism reduce the available material to minute proportions. This is true unless at the same time the observer increase his opportunities for observation beyond those usually sought even by such as are resident in a hospital. And, indeed, it is only when resident that the full opportunities of complete observation occur. Whenever a patient refuses food let the physician be made aware of it and let the food be preserved for his inspection. Let him see that his visits at night are made as silently as possible, and if it is necessary to use a naked light for more than a few moments in the examination of any patient let him screen that patient's bed, so that the remainder of the ward may not be illuminated. Let him learn the habits of his patients and cultivate the tradition that there is nothing too trivial or common for his interested attention.

(*A*) *Hallucinations of Vision.*

Before we enter upon a consideration of the various forms that may be assumed by hallucinations of vision in these cases let me give you an actual instance.

A man, aged 29,[1] who had been a painter, was admitted to the hospital with pulmonary tuberculosis. The disease affected both apices and there were signs of a previous pleurisy at the right base. He complained that occasionally during the nine months of his illness and more particularly during the few weeks preceding his admission he imagined he saw someone standing at the foot of his bed or walking across the room. Five days after admission he woke up to see a figure in a long cloak all in black at the foot of his bed. The face and arms seemed to be covered in the cloak. He was very frightened, shivered as if with cold, and sweated profusely. It seemed to upset him altogether and he lay awake with a bad headache, feeling depressed and ill.

The commonest form assumed by the hallucination of vision in these cases is that of a figure; neither arms nor legs are seen, and the figure appears to be draped and not clothed. Patients are unanimous that this figure is unlike anything they have ever seen and they are peculiarly definite in describing it as "draped," "wrapped in a shawl," "wrapped in a sheet," and in two instances volunteered that "it was dressed like statues are dressed and not in proper clothes." The limbs are never visible and some patients account for this by saying the hands and arms were hidden under a cloak. The head may be covered, but one patient said he saw long dark hair hanging on either side of the face. The face is frequently misty and it is probable that this mistiness gives rise to the impression that there is a "shawl on the head," or that the figure has "long black hair" on either side of the face.

Sometimes, however, it may consist of a face only, and one patient before admission was troubled by a face looking through the bars at the bottom of his bed. From this there is an easy transition to a "pair of eyes looking through the bars at the foot of the bed," of which more than one patient complained.

In one case only the patient saw a hand and arm come round the door of the ward that stood open flush with her bed. This arm was extended on to the white counterpane, and she sat up in bed expecting the body to appear round the edge of the wide-opened door.

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[1] Example 6, p. 419. (Case No. 68, table 3).

These hallucinations in all cases are white, black, or grey. They are never, so far as my experience goes, coloured or even normally tinted. For if the face is white, even the lips are said to be colourless, and it is probably this peculiarity that causes patients to describe such hallucinations as "the figure or face of a corpse." This forms a differentiating point between these hallucinations of the sane and the usual hallucinations of the insane. For in the insane the vast majority of hallucinations of vision either resemble some normal object or are highly coloured. I do not wish it to be understood that these black or white hallucinations do not occur to the insane; but they only form a small proportion of the total hallucinations of vision that occur amongst patients in an asylum. On the other hand, when delirium is absent, hallucinations of vision in sane persons suffering from visceral disease are always of the type I have just described.[1]

Patients express great difficulty in deciding whether the figure was that of a man or a woman. The face is frequently not visible. When seen it is always beardless, which makes many say they think it must have been a woman. On the other hand, the complete flatness and absence of the usual undulations of a woman's figure, suggest that it may have been that of a man. If the figure was white, most say at once "It was a corpse;" if it was dark, most deny the possibility of telling whether it were male or female.

Curiously, not one of my patients spoke of the hallucination as a ghost, possibly owing to the almost entire ignorance shown by the London-bred population of ghost stories or fairy tales.

Whatever the form assumed, the figure or face is single. I have found no instance so far in the sane where more than one face or figure appeared if the patient's eyes were open. I have very carefully excluded the not uncommon condition where faces or figures seem to stream past when the eyes are closed, though the patient is awake.

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[1] I have carefully excluded all cases where alcoholic excess might be expected.

In the majority of instances the figure remained stationary at the foot or on one side or other of the bed. It may, however, pass in front of the bed from left to right or from right to left, and is sometimes seen in the doorway. If it assume the form of a single face it is usually seen at the foot of the bed, and not uncommonly is said to "grimace." Usually the patient wakes from sleep to see it standing near his bed, and after a variable interval it disappears without movement. On the appearance of such an hallucination several male patients, before admission to the hospital, sprang out of bed to seize the person whom they supposed to have entered the room. The figure at once disappeared.

No sound accompanied the appearance of the hallucination of vision in any case; the figure did not speak, and when it moved no footsteps were heard. It made no gestures, and the "grimacing" occasionally mentioned probably refers rather to an unpleasant expression of the face than to any mobility of its features.

Such hallucinations of vision do not occur in the bright light of day. Usually the patient wakes from sleep to see the figure or face beside his bed. But in two cases,[1] although the hallucination occurred at night the patient was lying awake and saw the figure appear. Another patient[2] saw a figure in the corridor of the hospital when she got out of bed early on a February morning. In other cases the hallucination appeared in the evening when the patient was sitting quietly in a dimly lighted room.

Almost every case felt frightened with the first hallucination of vision, and in many the fear was accompanied by sweating, heart-beating, and "goose-flesh." If the hallucination is frequently repeated men, as a rule, recover their self-control and ultimately come to look upon the repetition of the hallucination with curiosity, often accompanied by anger that they should be "so fooled."

These hallucinations are not uncommonly accompanied by the depressed mood I shall describe later. In such cases the patient is convinced that the appearance is a sign that some ill-fortune has happened, or is about to happen. He believes some near relative is dead or in trouble, and not infrequently says that the figure was that of some dead relation, *e.g.*, "my sister." Asked if it resembled his sister he answered, "Not in the least, but I know it must have been my sister because she is the only one of my relations who is dead."

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[1] No. 9, table 1, and No. 90, table 3.

[2] No. 79, table 3.

The feeling-tone that accompanies the colourless white figure varies greatly, but the dark or black hallucinations are uniformly associated with fear or its physical manifestations.

[Amongst the cases reported in full instances of hallucinations of vision will be found in example 1, p. 404, § 3; example 4, p. 415, § 3; example 6, p. 420, § 4.]

(*B*) *Hallucinations of Hearing.*

All these hallucinations are of a simple type and in this way resemble the hallucinations of vision that occur under similar conditions. Owing to this characteristic it is more difficult to establish their independence of an external cause than is the case with the more complex auditory hallucinations amongst the insane. For if an insane patient complains that when he is alone at night a voice cries out to him that he is going to hell, it is certain that he is the subject of an hallucination of hearing; but if a sane patient states that before admission to the hospital when alone at night he heard knocking on the wall by his bedside it is impossible to be certain that the sensation had no external basis. Even when the patient is under continuous observation in a hospital it is frequently impossible to prove that the sound he says he has heard is an hallucination. But in the following case I was not only present myself during more than one hallucination of hearing, but was also able to demonstrate the associated conditions to a colleague.

The patient, a bright, healthy young woman, aged 23, was undergoing the training of a nurse. She returned from a short holiday with a temperature of 100° F., was warded for three days and then, as the temperature had fallen, returned to duty. Four days after she was again warded with slight swelling of knees and ankles which passed away rapidly. Then she began to suffer from reflected pains in the lower part of the chest accompanied by superficial tenderness, mainly over the seventh and eighth dorsal areas on the left side. She complained of temporal and vertical headache which was also associated with superficial tenderness. She was short of breath on the least exertion and the temperature continued to rise occasionally to 100° F. Ultimately a systolic murmur appeared at the apex which was not conducted to the back and did not obscure the first sound. The pulmonary second sound was accentuated. No other system showed any abnormal signs. The reflected pain and the temporal and vertical headache caused her considerable discomfort and for the first time in her life she began to suffer from paroxysmal attacks of depression. She also complained of curious noises under her bed at night. One night at 11.30 p.m. I passed her door and noticed that she was sitting bolt upright in bed with the gas blazing to its full extent; she looked frightened and was sweating. Her temperature was 99° F. She had a good deal of pain and tenderness over the eighth dorsal area on both sides, greater on the left than on the right side. There was marked superficial tenderness over the vertical area of the scalp on both sides. No other fresh physical signs had made their appearance.

She then told me she had not been able to sleep because she had been troubled by odd sounds and that at the moment she could still hear a noise resembling loud breathing or inarticulate whispering under her bed. For this I was able to assure myself there was no external cause. Her hearing was good (watch heard at three feet left and right), the meatus was free from wax, the membranes were normal and showed no cupping.

These noises occurred repeatedly at varying intervals during the next two weeks, and my colleague and I were able to assure ourselves that these sounds were truly hallucinatory and to study the conditions under which they made their appearance. She was not taking salicylic acid or quinine in any form.

The last traces of pain left her a month afterwards and up till her marriage, six months later, she had no further hallucinations. The systolic murmur persisted over the mitral area.

Such hallucinations of hearing, occurring in sane persons suffering from visceral disease, are never articulate voices and in this way differ characteristically from the commonest form of hallucination of hearing amongst the insane. Amongst my cases the hallucinations assumed the form of tapping or knocking (eleven cases), the sound of one or more bells (seven cases), a low whistling sound (one case), a sound like treading or heavy footsteps (one case), and a sort of breathing or inarticulate whispering (one case).

It is not necessary to be present in order to recognise that some of the sounds heard by these patients during their stay in hospital are hallucinations. Thus one patient complained that she heard a noise as if someone was stamping or walking heavily in the small space that separated the left side of her bed from a thick internal wall. Again, it is clearly improbable that the sound of a small bell rung sharply in the middle of the ward at night was other than an hallucination.

And it is sometimes possible to be tolerably certain that some of the sounds which these people complain of having heard before admission to the hospital were hallucinatory—as, for instance, where a male patient, hearing continuous knocking outside his window, compelled his wife to go down to find out why they were mending the street in the middle of the night. Again,[1] one patient, who was the subject of a well-observed hallucination of hearing in hospital, told me that since her illness, when sitting alone in her kitchen at home, she has heard heavy footsteps come through the door behind and to the left of her seat. Finding no one had entered the room she, on several occasions, turned her chair round. Shortly after she would again hear the same heavy footsteps which, now that she was facing the door, appeared to come from the wall behind and to the left of her chair.

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[1] Case No. 14, table 1.

The intensity of such hallucinations of hearing is very variable. Sometimes they consist of a continuous sound such as a whistle, sometimes of a series of sounds of equal intensity (knocking and tapping). Sometimes the sound begins softly and works up to a bang, or it may even rise and fall in intensity. If the sound increases it is usually said to come nearer, if it decreases it seems to recede.

These hallucinations usually occur at night. But as it is only necessary for their appearance that disturbing noises shall be absent they may occur during the day if the patient is in a quiet place; for as light was found to be a disturbing factor in the case of visual hallucinations so external noise seems to prevent the appearance of auditory hallucinations. They always cease when the patient is spoken to, but may begin again when silence is re-established, as in the case of the patient cited above (p. 356).

The feeling-tone that accompanies these auditory hallucinations varies greatly in intensity, but is almost invariably unpleasant. Two patients only described their hallucinations as pleasant. The patient is usually frightened and this fear is usually associated with sweating, with "beating" of the heart and "goose-flesh." One very intelligent patient told me she was much more frightened by the tapping on the wall than if it had been real tapping.

If the sounds are frequently repeated the patient may overcome his fear and may make elaborate observations to discover whether the sounds have any external basis, observations that greatly add to the value of the account.[1]

In all cases these sounds have a curious insistent quality. The patient is unable to listen to anything else unless he be directly spoken to, nor can he distract his attention from the sound as would have been possible had it not been hallucinatory.

[For instances of hallucinations of hearing among the fully reported cases see example 3, p. 412, § 3; example 1, p. 405, § 4; example 2, p. 409, § 4.]

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[1] Example No. 1, p. 405, § 4.

(C) *Hallucinations of Smell.*

Hallucinations of smell are the most difficult to investigate, for it is obviously impossible to prove that a smell of which the patient complains has no external cause unless you are present at the time and able to examine his surroundings and the condition of his tongue, mouth and nose at the moment when he experienced the sensation.

But the following case[1] is a good instance of a complete observation. A man, aged 45, suffering with aneurism of the aorta, suddenly developed pain and tenderness over the fifth, sixth, seventh and eighth dorsal areas on the left side, accompanied by marked scalp tenderness over the temporal and vertical regions. The physical signs remained unaltered, but his cough increased. At three o'clock one afternoon I noticed he was sitting up, leaning forwards, evidently uncomfortable, and found he was complaining of a foul burning smell that made him feel sick, "as if someone were burning very foul tobacco or something of that kind." He said the mug of coffee and milk that stood by the bedside stank of it, and that it was all round him. His breath and the small amount of sputum he was bringing up were free from odour. His tongue was clear, and examination of nose and throat revealed no abnormality. His sense of smell was good, and he asked to be allowed to continue smelling the essence of peppermint because it seemed "to drive the foul smell away for a time and to be reviving." When he ceased to smell the peppermint bottle the hallucination again obtruded itself on his consciousness. Ammonia acted even more strongly in the same direction.

He complained that this smell made him feel sick, but he did not vomit. By 4.10 p.m. the whole had passed away and he was able to drink his coffee.

This observation contains all the typical features of an hallucination of smell as it occurs among sane persons suffering from visceral disease.

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[1] Case No. 7, table 1.

Such hallucinations are invariably unpleasant, and the various forms they assume may be classified under the following headings:—

(A) *The sulphuretted hydrogen group.*—Under this heading I have placed those hallucinations where the smell was said to resemble "drains," "rotten fish, eggs, or cheese," and "stale greens." The hallucination of smell belonged to this type in seventeen instances.

(B) *The burning group.*—This is a very well defined group, for the patients usually complain of a foul burning smell, or add some further characteristic like "burning bricks," "burning rags." They also complain that their food is "burnt" or "smoky." The hallucination belonged to this group in nine instances.

(C) "*Earthy smell*" *or* "*smell of the grave.*"—I do not know where to place this type, which occurs sufficiently often to make it worth mentioning. Patients deny that it is like a drain, and one thought it not only was around him but clung to his hands.[1]

(D) *The smell of gas.*—This is a peculiarly definite form, for patients will get up and go round with a taper to look for the escape of gas.

(E) *Fæcal.*—Only one patient complained that the smell was fæcal, saying that there was a pot of fæces under his bed in the hospital. But I suspect that either this case should go into group "A," or that several instances included in group "A" should be transferred to this heading.

Hallucinations of sight are prevented by light; those of sound by the noises of the day. In the same way those of smell seem to be checked by strong external scents. A strong external scent, such as that of peppermint, does not blend with the hallucination but drives it away.

As neither light nor sound prevent the occurrence of hallucinations of smell they appear readily in the daylight hours. Thus, although these hallucinations are more difficult to establish, the time of their occurrence makes them more amenable to rigid examination than the majority of hallucinations of vision and hearing.

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[1] One patient who was a jobbing gardener attempted to describe this form of hallucination of smell by saying "it is like bad soil being turned up from the ground," "as if you had put cut grass into a heap and then turned it over after wet weather."

These hallucinations of smell frequently appear in relation to food, and the patient may rise and leave the table, saying that the food stinks. But they are not uncommonly present apart from food and are projected on to and intensified by any food that may be offered.

Thus these hallucinations become a not uncommon cause of sudden apparently inexplicable refusal of food by a patient. Some patients have learnt by repeated experience that these smells "are only fancy" and will put aside the food that seems to stink to be eaten when the smell has passed away.

Nausea is an almost universal accompaniment of these hallucinations, and if food is taken may culminate in vomiting.

Thus such hallucinations of smell are uniformly associated with an unpleasant feeling-tone, but are not accompanied by fear, "goose-flesh," or increase in the rate of the heart's beat. Such patients not uncommonly sweat during the hallucination, but as sweating is a common accompaniment of the nausea that precedes vomiting I am unable to say whether it is associated with the hallucination or with this nausea.

[For instances of hallucinations of smell among the fully reported cases, see example 2, p. 408, § 3; example 3, p. 413, § 4; example 5, p. 418, § 7; example 7, p. 424, § 5.]

§ 2.—Moods.

(*A*) *Sense of Ill-being.*

All of us when ill, and particularly if we are suffering pain, tend to be miserable. We think of the work we should be doing and of all we might have done had we been well. Lying in bed is irksome, and thought chases thought in an intolerable round. Patients of the hospital class worry about the rent that has to be paid and the children at home to be fed.

When in this state we can give reasons for our depression, and it is a relief to talk over our troubles with a friend, and when he has gone we wonder how he has cheered us. Music is pleasant, and some general amusement, such as the theatre, will tend to remove the depression and "help to take us out of ourselves."

But the condition to which I wish to draw your attention as a frequent accompaniment of visceral disease differs markedly from the state I have just described. For it comes on in paroxysms without reason and forces the patient to seek some solitary place. If it attacks him during a meal the patient will rise and leave the table as if to vomit. He will sit in some obscure corner of the ward away from his fellows or will retire to his own room and shut himself in. He will leave his home and seek some solitary place in the fields where he may weep alone and unseen. The public-house and every public place is repulsive to him. If in bed in a large ward he will turn over and hide his face in the pillow or cover it with the sheet. He usually has an intense desire to weep—a desire that in many cases he is unable to control and yet he is unable to say why he is miserable or why he weeps.

To be spoken to only deepens his misery and many a patient who has controlled himself up to that moment will burst into tears when spoken to kindly. Thus as the sister goes her round in the wards it is not uncommon for some patient to avert his head and remain dumb to her questions, knowing that if he answers he will weep.[1]

Music is intensely displeasing to a patient in this state. The hospital patient who is troubled about his work or the support of his family will usually become cheerful again under the influence of some entertainment in the ward; but those who are suffering from the causeless depression I am attempting to bring to your notice become more miserable on such occasions, and I have seen them rise from their places and retire to their beds or to the furthest corner of the ward.

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[1] To prevent misunderstanding, I in no way suppose that this condition is peculiar to those suffering from visceral disease. It can make its appearance as a primary mental manifestation, and in association with organic or functional diseases of the nervous system. Such cases are, however, excluded from our present consideration and my object will be to describe the conditions under which this mental state may make its appearance in sane persons who suffer from visceral disease.

At the same time many are haunted by a vague idea of impending ill. They do not know the nature of this ill nor to whom it will happen. This leads to many curious acts. Thus a workman will pin his name and address inside his coat that he may be known in case of accident. Under the influence of this mood patients in hospital write home to ask what misfortune has happened or which of their relations is ill.

A considerable proportion of us are in the habit of visualising more or less strongly. That is to say, if most of us are asked to think of our home we are able readily to picture the details of some room, we see the colour of the walls, the table, the carpet, chairs, and other familiar belongings. Moreover, the room is either seen illuminated by sunshine or by lamplight, and not infrequently a fire is burning cheerfully in the grate. This faculty is not a prerogative of the educated and well-to-do, but is found widely amongst the more sober though ill-educated members of the working classes.

Now, whereas the normal man is incapable of visualising his home other than coloured, lighted and garnished, one who is in the mood I am describing is haunted by a picture that is in every way the reverse. The room is badly lighted, and often the windows are said to be dirty or sooty; the gay paper has gone from the walls which now seem gray, pictures and ornaments are gone, the grate is fireless, and the furniture in disorder. The room looks "as if we had been sold up," "as if there had been a row in it and everything had been thrown about," "as if it had been deserted and no one had been there for months." In one particularly marked case a patient with phthisis asked his wife why she had made such alterations in the home during his absence?[1]

When the depression has lasted a long while or has recurred frequently, these patients may have a vague desire that the end may come. But the majority spurn the idea of suicide, and in the very few cases that ever really entertained the idea the motor acts necessary for the fulfilment of the impulse immediately removed all desire for self-destruction. Thus, one woman with phthisis walked out to throw herself into the canal, but before she reached the bridge the impulse had entirely gone. A youth rose from his bed to reach some ferrocyanide of potassium on a shelf in his room; but the act of climbing on to the chair to reach it restored his self-control. It is, of course, possible that people in this condition may commit suicide and so never come to investigation. But I have met with no case where suicide was actually attempted unless other factors came into play in addition to the mood I have attempted to describe.[1]

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[1] Example 7, p. 423, § 4.

The duration of this mood may be extremely short. It may arise with great suddenness and pass away with equal rapidity in half an hour. But not infrequently attacks lasting an hour or more recur at frequent intervals throughout a day, to be repeated on the morrow, and the patient will then say his depression has lasted "one or two days." It is, however, not usually continuous throughout many days, but ebbs and flows in attacks of varying duration.

[Amongst the fully reported cases compare example 1, p. 405, § 6; example 2, p. 410, § 6; example 5, p. 417, § 4; example 7, p. 423, § 4; example 8, p. 425, § 3.]

(*B*) *Exaltation.*

But sometimes patients with disease of the heart or lungs are subject to an exactly opposite feeling of exaltation. It is in no sense a delusion but is a mood accompanied by a feeling of physical strength. A feeling suddenly comes over them that they are able to do even more than in the days before they were ill; but immediately any attempt is made to act on this feeling their weakness is brought home to them. Men under the influence of this exalted sense of well-being attempt to lift heavy weights, box with fellow patients, or to perform some equally impossible physical task. Women of the hospital class are seized with a passionate desire to wash and scrub, and in hospital will move beds or attempt to lift heavy pails.

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[1] Particularly alcohol.

Such efforts at once betray the patient's real weakness, and he will then go back to bed or lie down convinced that this sense of increased well-being is founded on no real basis of increased strength.

One patient told me that during this period of exaltation she could sit down and eat enough for two working men, and can enjoy everything she eats.

[Amongst the fully reported cases compare example 1, p. 406, § 7; example 2, p. 410, § 7; example 5, p. 417, § 5.]

§ 3. SUSPICION.

When the depression of which I have just spoken has been frequently repeated, or has lasted for a considerable time, an abnormal state of suspicion is liable to arise in the minds of these patients. They believe their friends "want to get rid of them," that their fellow workmen "want to get them the sack," that master, mistress, or superior officer think them "lazy and idle," that the medical officer, sister and nurses think they are shamming or not so ill as they seem to be.

Yet this suspicion is more an impulse than a formed delusion. If they see two people talking together who stand in any relation to them they imagine that these people are talking about them. But they always deny that they have overheard any words to support their suspicion. Under no circumstances do they imagine a definite charge of fraud, impiety, or immorality is made against them, but they imagine that one person is telling the other that they are lazy, idle, good for nothing, or shamming. So vivid is this suspicion that these patients frequently accuse their friends, fellow workmen or employers of desiring to get rid of them and when in hospital they will accuse the nurses of neglecting them.

And yet this suspicion is so directly the outcome of an emotional state, and so little a fixed delusion, that all the time they recognise it has no basis of logical truth. They will tell you that they know it is not true, but they have an "irresistible feeling" at the moment that it is so. The charge once made and met by a simple negative they are, as a rule, perfectly satisfied. On several occasions when the patient had previously been subject to this form of suspicion, and had made an ill-defined charge of neglect against the nurse, he has told me that he knew it was not true, but the "funny feeling" came over him and he was compelled to make the charge. Such patients are therefore glad to accept a simple denial and for the matter to be allowed to drop.

In this way they differ fundamentally from the majority of delusional cases amongst the insane. For the insane are firmly convinced of the logical truth of their suspicions; whereas the state of mind that I have attempted to describe is characterised by a sense of invasion by an impulse from without to think and say things that are contrary to reason.

Those who have learnt by repeated experience that this suspicion has as little basis of truth as the other "funny feelings" to which they have been subject since their illness came on, as a rule, control themselves sufficiently not to openly accuse those around them of wishing them ill; but before this condition is reached much unhappiness is frequently caused, for it is always concerning those nearest to them and not concerning strangers or casual acquaintances that this suspicion makes itself felt.

[Amongst the fully reported cases compare example 1, p. 406, § 10; example 2, p. 410, § 9; example 5, p. 418, § 11; example 9, p. 428, § 7.]

CHAPTER II.—THE CAUSES THAT UNDERLIE THESE PHENOMENA.

I HAVE attempted to show that the most various diseases of internal organs are liable to be accompanied by changes in the mental state of otherwise sane persons. It now remains for us to find the common factor in such diseases that is associated with the presence of these mental changes.

For this purpose I have selected from my records cases of disease of the lungs and of the heart and have divided them into two groups according to whether they showed these mental changes or not. From the one group where these changes were present I have constructed tables 1 and 3; from the other where they were absent tables 2 and 4. No case has been included unless it has been under my own observation for at least five weeks.

EXPLANATION OF THE TABLES.

Throughout these tables—

+ Signifies that the mental change indicated made its appearance when the patient was not in hospital.

X Signifies that it occurred when the patient was under my observation in hospital; but that my attention was not called to its occurrence until more than 24 hours afterwards.

X Signifies that I was present during the occurrence, or that I made a complete examination of the patient's physical condition, skin sensation, special senses, and, in the case of an hallucination, into possible external causes within 24 hours of the event.

The area occupied by the superficial tenderness that was present in each case is given as "Cervical 8," "D. 7," &c. By this I do not mean to imply that Cervical 8 or Dorsal 7 were marked out to their complete extent, but only that more or less tenderness was present within the territory supplied by these visceral sensory segments. Again, "D. 7, 8, 9, 10" is only intended to signify that tenderness was widely distributed within an area bounded above by the usual upper border of Dorsal 7 and below by the lower border of Dorsal 10.

In each case of mitral disease the physical signs at the apex are given in addition to the usual headings—"Mitral Stenosis," or "Mitral Stenosis and Regurgitation." In every case where aortic disease was said to be accompanied by mitral regurgitation it is understood that the 1st sound at the apex of the heart was abolished and the systolic murmur conducted to some point in the axilla or back external to the apex beat.[1]

The numbers in the columns under wasting refer to the gain or loss of weight during the patient's stay in hospital.

On these charts I have not been able to include all the facts observed in each case which might have been supposed to stand in some relation to the mental changes I have described. Nor have I been able to indicate the changes undergone by several of the cases during the many months they were under observation. But it is at once obvious on comparing tables 1 and 2, or 3 and 4, that mental changes are not confined to any one form of disease.

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[1] *Cf.* "Pain in Diseases of the Heart and Lungs," BRAIN, 1896, p. 183.

Nor can fever be the determining factor in their appearance, for many of the cardiac cases that showed the most marked mental changes were afebrile, and there are as many cases of lung disease with more or less fever on table 4 as on table 3.

Loss of weight cannot be the determining cause of their appearance, for most of the cardiac cases with mental changes had not lost weight, and there are as many cases of lung disease on table 4 that have lost weight as on table 3.

No vascular disturbance will account for their presence, for many of the cases in which compensation was most disturbed showed no mental change.[1]

Nor can they be accounted for by anæmia, for the profoundest anæmias may be entirely free from any of the changes I have described, although they may be well marked in a simple case of chlorosis. Thus out of thirteen cases of simple anæmia five suffered from no pain or tenderness, and were free from any of these mental changes. In the remaining eight, pain and headache, accompanied by more or less superficial tenderness were present, and in these cases one or more of the mental changes I have described made its appearance. Out of eight cases of pernicious anæmia, all of which presented the characteristic clinical appearance and typical blood changes of the disease, seven were entirely free from pain and tenderness, and from any of the mental changes I have so far described. In one only did they make their appearance, and in this case they seemed to be associated with the development of pain of gastro-intestinal origin.

The only factor that is universally present on tables 1 and 3 and absent from tables 2 and 4 is reflected visceral pain.

This type of pain is produced by impulses passing from the affected organ up the fibres of the sympathetic, through

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[1] I have been careful to exclude all cases of so-called cardiac delirium which not infrequently accompanies a failing heart; for this psychosis belongs to a different category.

the ganglion of the posterior root, into the central nervous system. A disturbance is thereby set up in those segments that stand in relation with the affected organ. Other sensory impressions entering the same segments are modified, and pain is produced radiating round the body, accompanied by increased sensibility to pain of the superficial structures of the body within the supply of the affected segments.

But it must not be supposed that every case that suffers from visceral reflected pain and its accompanying superficial tenderness must therefore immediately show mental changes. Such an idea would be negatived by the experience of anyone who has suffered from a toothache.

The pain must be of considerable intensity and of some duration. I have prepared a table showing those cases of pulmonary tuberculosis in which the pain was either of small extent, slight intensity or short duration (table 5). It will be seen that although none of these cases suffered from any mental change during their six weeks stay in hospital many of them had experienced one or more of these symptoms before admission. Thus we find that whereas the cases which had at no time suffered from pain were entirely free from these mental changes, such of them as showed some pain either of slight extent or short duration had exhibited some of these phenomena before admission, although they remained throughout their stay in hospital free from any such change.

There are many cases in which the patient has been admitted free from all mental changes but has developed them in association with extension of the disease during his stay in hospital. It was then possible to watch the development or disappearance of the mental changes with the coincident appearance or disappearance of reflected visceral pain and tenderness.[1]

But apart from the fact that where these mental changes were present amongst cases of disease of the heart and lungs pain was a universal concomitant, the tables I have put together tend to the same conclusion by a somewhat different method of reasoning.

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[1] Compare example 4, p. 415, § 3 and 4.

For, as I have shown in a previous paper,[1] there are certain conditions, more particularly in cardiac disease, in which reflected visceral pain is usually absent. For instance, I have shown that when in disease of the mitral valves a systolic murmur heard over apex of the heart is conducted to the back and abolishes the first sound over the apex, the axilla and the back, no reflected pain is usually present. Now all the cases[2] that presented these signs are found on table 2, the table that includes those patients who suffered from no mental changes.

But on the other hand, where a systolic murmur is heard over the mitral area accompanied by a short, sharp first sound, with or without a murmur of mitral stenosis, pain will tend to be a feature of the case. Such cases[3] will be found to fall into table 1, which comprises those patients who suffered from some one of the mental changes I have described.

Again, in cases of aortic regurgitation where marked signs of mitral regurgitation are present and the first sound is abolished at the apex of the heart, the characteristic pain of aortic disease is usually absent. Such cases[4] also tend to fall into table 2, which is composed of cases without mental changes.

In a similar way the greater the extent to which the progress of tubercular phthisis tends to excavation and rapid destruction of lung tissue, the less will these mental phenomena tend to be a feature in the case. For rapid excavation of the lungs destroys the nerve endings and thus prevents the production of reflected pain.

Local pain, such as produced by a pleurisy, is not, as far as my experience goes at present, usually associated with these mental phenomena. Thus amongst nine cases of pleurisy in which the pain was of varying severity, not one showed any of the mental phenomena I have described.

Now it might be urged that the pain of a pleurisy is not intense enough to evoke these mental phenomena. But although there is probably much truth in this contention I incline at present to the view that there is in this matter a fundamental difference between the two types of pain; for just as a local pain of somatic origin, such as that of a burn, is unaccompanied by these mental changes, so an equally local pain produced by an inflammation of the inner lining of the body wall will also be unassociated with these phenomena.

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[1] "On Pain in Diseases of the Heart and Lungs," BRAIN, 1896.

[2] Nos. 42 to 48. [3] Nos. 13 to 22. [4] Nos. 28 to 37.

On the other hand, reflected pain, although accompanied by tenderness of the body wall, is of visceral origin. It is not surprising, therefore, that two types of pain so fundamentally different, should be associated, on the one hand with the presence, on the the other with the absence, of these mental changes.

But although reflected visceral pain seems to be the one universal concomitant of these mental changes in cases of visceral disease amongst sane persons, other factors undoubtedly predispose to their appearance.

Sex plays a considerable part in the facility with which they occur. For three times as many women as men had hallucinations during their stay in hospital, although the total number of cases used in this research comprised 102 men and 90 women.

If tables 1 and 2 are examined it will be seen that of sixty-five male cases with disease of the heart or lungs twenty-one showed one or more of these mental changes; whilst of sixty-one female cases thirty-four were affected in a similar manner.

Menstruation tends to favour the appearance of these symptoms, not only owing to the mental changes it produces directly, but also by its effect on visceral reflected pain. For menstruation (1) is in itself a cause of reflected pain; (2) it increases visceral reflected pain of whatever origin and causes it to spread widely; (3) it so lowers the resistance of the central nervous system that visceral reflected pain makes its appearance where it would not otherwise do so. I have therefore refrained from including in my results any phenomena that occurred during the menstrual period or during two days on either side of this period. In every case a strict record was kept upon the temperature chart of the time and duration of menstruation, and this period avoided in all subsequent compilation. But even so, the full mental effects of menstruation cannot be eliminated, and the fact that a greater number of women than men showed these mental changes must be put down in part to the predisposing influence of menstruation.

Fever also favours the production of these mental changes, not only by decreasing the resistance, but also by causing wide-spread generalisation of any reflected pain that may be already present. Thus one case developed marked reflected pain and superficial tenderness with a sudden rise of temperature that lasted two days. During this period she experienced her first hallucination. But here, as in all similar cases, we have to deal not only with the effect of the fever as such on the mental state, but also its indirect effect due to the sharp increase in the visceral reflected pain that it may produce. This is probably one of the reasons why pulmonary tuberculosis is more commonly associated with these mental changes than cardiac disease. For most cases of phthisis are febrile, and the daily rise of temperature tends to cause an otherwise localised outburst of reflected pain to spread widely.

Anæmia also predisposes to the appearance of these phenomena, not only directly but also by favouring the appearance of reflected pain in answer to very slight visceral disturbances. But the severest cases of anæmia, pernicious anæmia and allied conditions, do not, as a rule, suffer from reflected pain, and the majority remain free from the mental changes described above. But in the last stages many of them develop a form of delirium which bears no relation to the phenomena under discussion.

Loss of weight, especially when rapid, seems to predispose to the appearance of these mental changes, but in no way determines their appearance. But in the last stages of tubercular disease, when profound wasting has taken place, a slight dementia may come on characterised by extreme loss of memory and attention. This state seems to be unfavourable to the development of the changes that form the subject of these lectures, which require a mind untouched by the lethargy that accompanies extreme wasting.

How far an hereditary taint of insanity plays a part in the production of these phenomena I am unable to say, as I have rigidly excluded all cases in which there was an heredity of insanity or epilepsy.

§ 1.—THE CAUSES THAT UNDERLIE THE DEPRESSED MOOD.

Up to the present I have attempted to show that whatever accessory factors may play a part in the production of these phenomena the main factor is the presence of reflected visceral pain accompanied by superficial tenderness.

It now remains to consider each symptom in detail to discover if possible the relation in which each of them stands to such reflected pain.

When describing the various symptoms that went to make up these mental changes, I began with the hallucinations because they were the most obvious deviation from the normal. Now, however, I shall begin with an analysis of the depressed mood, because its relation to reflected visceral pain is in some ways clearer than that of any other of these phenomena.

This depression is in reality an acute alteration of feeling-tone in the direction of ill-being. It is in no sense an intellectual state induced by the thoughts and worry engendered in the patient's mind by the illness from which he suffers. When depressed from such causes the patient may be dull and not want to speak, but can be cheered by a quiet word or by the visit of a friend. He rarely has the desire to cry, and the ill that he expects is well defined; he fears for his work or for those who are dependent on him. Intelligent patients who have experienced both forms of depression are emphatic in their expression of the difference between the two.

Nor can we suppose that this ill-being is part of that general depression which accompanies all pain, for then it would appear with other forms of pain than the reflected pain of visceral disease. One who suffers from a burn, or from an aching tooth, may be depressed and miserable, because in one case every movement, in the other, the taking of food, is accompanied by an increase of pain. But the misery so induced differs from the mood which makes up so important a part of the phenomena under discussion.

This mood may make its appearance apart from reflected visceral pain as a light form of true melancholia, and also in some neurasthenic states. But we are here dealing only with sane persons, and with such as are suffering from organic visceral disease.

But even sane persons who suffer from no pain are liable to waves of causeless depression. This depression, however, does not, in my experience, come on with the suddenness nor assume the intensity of the mood under discussion. Moreover, music is usually pleasant, the presence of a friend comforting, and a visit to some place of amusement will remove the last traces of this form of depression.

The most marked characteristics of the depression I have described are all associated with a sense that it lies outside reason. It comes and goes causelessly as far as the patient is concerned. It is an obsession dominating his rational life and producing feelings that he cannot understand. He has a sense of impending ill, but has no idea what it is, from what quarter it will come, or whom it will affect. All he feels is that it will affect him in some way to his hurt. He cries, but he does not know why he weeps, nor can he understand how he, who has never wept over serious losses, should now weep for no reason at all. He has a feeling that he has somehow lost control of his mental processes but that his reasoning powers are still intact. He never has the feeling that so frequently makes its appearance in the early stage of an insanity proper—that he is "losing his reason." Thus, when discussing his symptoms, he recognises that something of which he is ignorant has caused him to lose control, and that the results of this loss of control have been the appearance of baseless misery and baseless fears.

This feeling of ill-being is in no way an emotion proper, for its main characteristic is the absence of projection. In anger we are angry at something or with somebody, and it is very doubtful if we are capable of anger without projecting it on to an object. This projection I believe to be a necessary part of every true emotion, such as anger, love, hate, and the like.

But on the other hand, in the state I have attempted to describe, projection is absent or so slight that it is doubtful if, when it occurs, it is not a secondary change. For such a state we require a word in English pyschological terminology. I propose, then, to use the word "mood" to indicate a state of mind in which consciousness is dominated by feeling-tone but where the resulting state is not projected.[1]

But in order that this mood may be produced the pain must be of considerable intensity or duration, or must occur frequently. It is not sufficient that a patient should suffer from even considerable pain of the reflected visceral type if he has been healthy up to the time of its origin. The oftener visceral pain has appeared the more easily will its reappearance be accompanied by the mental changes I have described.

But not only are the duration and intensity of the pain of importance, but also the number of segmental areas affected. For the greater the area involved in pain and superficial tenderness the more certainly will mental changes make their appearance.

Again it would appear that the presence of pain and tenderness over some of these segmental areas is more likely to be accompanied by the sense of ill-being, than where the pain and tenderness affects other areas. Thus pain and tenderness over any of the abdominal areas is peculiarly liable to be associated with this sense of ill-being, whilst pain and tenderness over the upper thoracic areas must be relatively severer and of longer duration before it becomes associated with depression. A disease that causes reflected pain over the upper thoracic areas, such as uncomplicated aortic disease, is less likely therefore to be associated with depression than mitral stenosis with regurgitation, which is under certain conditions liable to produce pain and tenderness over the seventh, eighth, and ninth thoracic areas. For the same reason acute disease of the upper lobe of the lung is less likely to be accompanied by depression than affection of the lower lobe. For affections of the lower lobe are also liable to be accompanied by pain and tenderness over the seventh, eighth and ninth dorsal areas.

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[1] The best example of the difference between a mood and an emotion is given by the two types of fear. In fear, an emotion, we are afraid of a thing. But there is no doubt that a state of fear can exist in which there is no fear of any particular object. This condition, which is common in the insane and in children, is a true mood. I believe it can also exist in visceral disease, but is usually so closely mixed up with hallucinations or other manifestations that it becomes peculiarly difficult to investigate.

But these areas are peculiarly associated with gastric disturbances, and for this reason painful affections of the stomach are most frequently of all accompanied by this form of depression.

Many a case of phthisis will remain entirely free from depression until a sudden fresh affection of one lower lobe, or the onset of some gastro-intestinal complication causes a sudden outburst of pain and tenderness over some of these lower dorsal areas.

In the same way a case of chlorosis may remain entirely free from all mental changes until reflected pain makes its appearance in consequence of some gastro-intestinal irregularity.

But headache alone may be accompanied by this condition, if sufficiently prolonged and sufficiently severe. Thus the intense pain of herpes zoster ophthalmicus may be accompanied by typical depression, as I shall show later when discussing the causation of hallucinations. But I am inclined to think that headache of the reflected type, when unaccompanied by similar pain and tenderness over the trunk, is much less likely to be accompanied by depression than if the pain and tenderness were situated over the abdominal areas.

Thus, in order that this depression may make its appearance, it is necessary that the pain present should have been of this visceral reflected type; it must have lasted a sufficient time, and have reached a certain intensity. The time and intensity required depend in any one case on several accessory circumstances, such as the sex of the patient, the presence of anæmia or fever, and a certain inherent resistance natural to the person affected.

§ 2.—The Causes that Underlie the Feeling of Physical Exaltation.

But in addition to a depression I described a curious sense of physical exaltation that is present in some of these cases. It is not so universal as the depression, and, as it manifests itself only in a sense of well-being, is much less easy to investigate.

It is accompanied by no actual increase in physical strength, for a patient who gets up from his bed intending to clean the house down, is obliged to return to it, overcome by the effort of putting on his clothes.

However, it is not in any sense a delusion of grandeur, for the patient does not imagine he is peculiarly gifted intellectually or in relation to his surroundings. He does not believe he belongs, or should belong, to any social rank other than his own. He is always himself, but he is himself young and strong again. He feels the sense of well-being that exists in the normal man exaggerated. This state is therefore the exact converse of that sense of ill-being which made up the depressed mood.

Now there is much reason to believe that the more definitely a sensation is projected, the less is likely to be the feeling-tone by which it is accompanied. Thus the senses of smell and taste in which external projection is slight are accompanied by more feeling-tone than the senses of sight, touch and hearing. In the same way the ill-defined sensations such as those of the viscera come to consist in consciousness of little more than a feeling of well- or of ill-being.

Visceral pain will intensify this sense or ill-being, and produce the pathological state I have described as the depressed mood. If the pain diminishes and the depression passes away rapidly, a rapid reaction will take place. The patient will swing over into a condition not of normal but of exaggerated well-being.

If this explanation for the exaltation be correct it should stand in close relation to the cessation of the depression. This is so in a large number of cases. Thus in example 5, p. 417, § 5, it appeared under the following conditions. All the afternoon he had had pain in the head and chest, and during this time he had a slight attack of depression. The pain ceased at five o'clock, and then there came over him a feeling of physical strength and well-being. It came on quite suddenly and lasted about an hour. He began to make his bed, feeling he wanted to lift it, or something equally heavy. Then he boxed with a neighbouring patient. This taught him how weak he was, and he went back again to bed.

But although the exaltation occurs in its most complete form shortly after the sudden disappearance of an attack of depression, it will make its appearance if the pain suddenly improves, even if that pain has not on that occasion been accompanied by a definite attack of depression.

But apart from an attack of depression it is probable that a sense of exalted physical well-being can arise in another way. Within the space of each day the underlying feeling tone varies even in absolutely normal people. These variations are, however, so slight, and are so repeatedly covered by frequent alterations in the conditions of life that they escape notice. But to those of monotonously regular habits these variations throughout the day are appreciable.

Now this tone probably depends on the activity of the viscera, an activity that takes place outside consciousness, excepting for the feeling-tone it engenders.

Pain of the reflected type calls up the whole field of visceral activity into consciousness, and it is probable that those who are the subjects of these mental changes are thereby rendered more susceptible to normal alterations in visceral feeling-tone which would otherwise have passed unobserved. This would seem to be one cause of the exalted sense of well-being that sometimes appears in cases of visceral disease and accounts for the fact that this form of exaltation not uncommonly makes its appearance in the morning when the patient rises from bed.

The duration of this condition is generally short, but it probably helps to furnish instances of the well-recognised hopefulness of those suffering from phthisis. But, strictly speaking, the "spes phthisica" is a more permanent mental condition of a different origin and nature. Many a case of tuberculosis is practically ignorant of his condition. For he suffers from no pain. His cough is not more troublesome than it has been for a long while. He knows nothing of his temperature and of the physical signs of profound disease that have such a deadly significance for those around him. His is a condition of nescience, and when he talks of recovery, of returning to his business, we, who know how gravely the lungs are destroyed, wonder at his hopefulness.

This hopefulness, except in those cases that experience the feeling of physical exaltation, is based simply on ignorance of the increasing gravity of the signs of disease. If such a patient enters the hospital he quickly learns the significance of the fever indicated on the temperature chart that hangs above his head. The weekly weighing reveals to him his loss of weight. From the words or actions of doctors or students he gathers that they recognise marked signs of disease within his chest of which he is entirely ignorant. All these factors serve to destroy his exaggerated hopes, and many cases that enter a hospital hopeful to an extent that appears almost to amount to a delusion, leave it with a just sense of their condition.

Take, for instance, a man of 54, suffering from advanced tuberculosis of both lungs, with extreme signs of cavitation over the upper half of the left and consolidation of the upper lobe on the right side. His temperature lay mostly between 101° and 102°. He was extremely intelligent and particularly fond of reading. He had no pain and very little cough. He was emaciated, but the loss of weight had been so gradual it had not troubled him. On admission to the hospital he was hopeful and talked about returning to work; but when he went out, though still smiling, he had lost his exaggerated hopefulness. He said, "I know I shall not get well. I am losing weight and my temperature is always bad. I am going to make myself comfortable at home. I have arranged my sofa so that I can see to read by day and a gas jet so that I can read when it gets dark, and I think I shall spend a very pleasant three weeks—for that's about the time I think I shall live." Three weeks and two days after discharge he died quietly, reading his books up to the last.

Spes phthisica, when not a manifestation of physical exaltation, seems then to be based on a failure to comprehend the gravity of the disease—a failure due to the patient having no symptom that will serve to show him how rapidly progressive are the physical signs of disease.

For this reason cases of tuberculosis in which the larynx is the main focus of disease are, as far as my experience goes, mostly melancholic.[1] They do not as a rule suffer from much pain of the visceral reflected type, and are therefore free from the paroxysmal attacks of causeless depression with a sense of unknown ill impending. But with an incessant cough and progressive loss of voice they early realise the seriousness of their condition. Take as an instance the following case:—A man of 24 had been ill for two years with cough and loss of weight. He first lost his voice eighteen months before I saw him. On admission there were signs of phthisis at both apices, and an old pleurisy at the left base. His voice was reedy and the larynx showed the typical tubercular changes. His temperature rose to 101° every night. He was entirely free from reflected visceral pain or headache. He was frequently depressed, and felt as if he was too ill to do much. He sat still and said nothing, but liked people to talk to him if it did not involve his answering, and much enjoyed being read to. He constantly worried lest his voice should never be well enough for him to return to work. He had no feeling of impending ill or desire to cry. He much enjoyed an entertainment consisting of music and songs that took place during his stay in hospital, and played well at draughts, feeling happier if he could win a hard game.

There is, however, another condition which must not be confused with the sense of exaggerated physical well-being that I have described. Many cases of aortic disease seem to be liable to attacks of excitement, in which they flush. Then they may burst out into a paroxysm of anger or uproarious excitement. Occasionally during such attacks the men become violent and will strike another patient. One male patient with aortic disease told me he frequently had a feeling that he must destroy something; he fought against it, but could not control himself, and had pushed all the knives off the table so that he might not be tempted to use them against one of his children; his wife then recognised his condition and sent the children away. He was terrified lest the temptation should overcome him some day to do bodily harm to one of his children.[1]

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[1] *Cf* cases 126, 127, 128, and 130, table 4.

Again, let us take the following case of a young woman of 19 years of age, suffering from disease of the aortic valves without implication of the mitral as a sequel to several attacks of rheumatism. A double aortic murmur of the usual type was audible, abolishing both first and second sounds over the aortic area. The first sound in the left axilla was long and booming. The pulse was characteristically regurgitant.

She was a bright, cheerful girl, quick in her movements, and when there was no one to check her would run about the wards. At times she became extremely excitable. For instance, if, when she was washing up plates, another patient said anything that annoyed her, her face flushed violently, and she said what first came into her head. Occasionally her nose bled during this excitement. She always felt ill afterwards, and generally had to go to bed. Occasionally she would break out into wild fits of laughter, in which she could not stop. These also were followed by a feeling of illness.

This condition seemed to bear no relation to the reflected visceral pain from which the patient also suffered occasionally.

Thus in conclusion it would seem that the exalted sense of well-being, to which persons with visceral disease are liable, may arise from many different causes.

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[1] Case 9, table 1.

(1) It may be the direct contrast to a mood of ill-being induced by visceral pain.

(2) Those who suffer from visceral pain are liable to an accentuation of the normal waves of well- and ill-being that apparently stand in connection with the feeling-tone connected with visceral activity.

(3) The apparent exaltation may be due to ignorance on the part of the patient of the gravity of his condition. To this group belong many of the cases of so-called "spes phthisica."

(4) Patients suffering from disease of the aortic valves are liable to attacks of excitement apparently of vascular origin.

§ 3.—THE CAUSES THAT UNDERLIE THE STATE OF SUSPICION.

I pointed out that patients with visceral disease not uncommonly suffer from an ill-formed state of suspicion. They imagine their friends want to get rid of them, or are talking about them, and that the nurses wish them no good.

Comparison of a number of cases of cardiac and pulmonary disease showed that this symptom belonged to the group of mental changes that accompany pain of the reflected visceral type, for it is entirely absent in that group (tables 2 and 4) in which all pain of this kind was absent.

But unlike the depression it probably seldom, if ever, appears alone. In every case I have so far come across it was accompanied or preceded by the depression, and if tables 1 and 3 are examined it will be seen that there are many cases, where although other mental changes are well marked, this suspicion had not made its appearance.

It seems to take its origin directly out of the state of ill-being or depression; for one of the most marked features of this state of ill-being is the sense of impending ill. But the patient is ignorant what this ill may be, and on whom it will fall, except that in some way it will affect him. With this is associated a sense of his physical worthlessness. From this state it is but a small step to the idea that those around think meanly of him, that they believe he is not so ill as he makes out, that he is lazy. Seeing two persons talking together, who are associated with him either at his work or by friendship, he will think they are talking about him, and if about him they will surely be telling one another what he believes they think. They will say he is lazy and that he must be got rid of from his work.

Such patients have no feeling of moral unworthiness, and therefore, when a prey to suspicion, they have no delusion that they have committed a crime or a sin, or are in any way shunned by their fellows.

Thus the form assumed by the suspicion in these cases of visceral disease among the sane is fundamentally different from the commonest form of suspicion amongst the insane. For the insane patient thinks that those around him believe he has committed some act either against religion, the law, or social custom. He believes that every passer-by in the street thinks he has profaned the mass, poisoned his father, or acted as a blackleg in his trade.

Again, another difference comes out between the two states when an attempt is made to reason with the patient, for the insane patient will pile up reasons to show that his belief is true. "The last time he went to church no one would sit near him;" the policeman up his street is "always smelling about his place;" the workmen on his job "won't speak a word more than they are obliged" to him. Point out to such a patient that these beliefs are incorrect, or bring overpowering arguments to prove their falsity, and he will leave you silenced but not convinced.

On the other hand the same patient with suspicion born out of a sense of ill-being of visceral origin is never certain for long together that people are against him. The idea comes and goes in such an inconsequent manner that even when he keeps it entirely to himself he is frequently not quite certain of its truth. If, however, he should make a complaint to his relations, friends, fellow-workmen, master, or to some hospital authority, a simple negative entirely satisfies him. When such patients have complained to me about the way some nurse is against them I have often asked, "Do you believe what you say is true?" Frequently they will answer that they don't know what to think. At one moment it seems true, and at another false. Some of the most intelligent seem to look upon it as something that seizes them from without, as if their reason were invaded by something over which they had no control.

No reasons are necessary to prove to these patients that what they hold is false. A simple denial will satisfy them. It matters little whether this denial comes from the person whom they believe to be against them or from some superior not implicated in the suspicion, such as their employer or physician. It is as if a definite authoritative statement from without were enough to enable their reason to gain the upper hand of this non-rational belief. Thus, usually, they express themselves completely satisfied with a simple denial, in extreme contrast to the insane who, though compelled by the weight of arguments to silence, are still unsatisfied.

In its causeless or inconsequent onset, and in the sense of loss of control by which it is accompanied, it closely resembles the state of ill-being out of which I have tried to show that it springs.

§ 4.—WHAT IS THE DETERMINING FACTOR UNDERLYING THE APPEARANCE OF HALLUCINATIONS?

We have seen that, although there are many predisposing factors, the one necessary concomitant of these phenomena is pain of the reflected visceral type. It will now be well to consider whether we can differentiate further the causes underlying the appearance of the hallucinations that form so important a part of this mental state.

For instance, are we to consider that one or more hallucinations will necessarily appear because the patient has become subject to paroxysmal attacks of depression and suspicion; or is there some other determining factor necessary before hallucinations can make their appearance? For, as I have already shown, the exaltation and suspicion may in many cases be deduced from a primary depression. But the hallucinations seem to stand in no such relation. They occur in a comparatively small proportion of the cases in which the other symptoms are well marked. In no case do they appear without the depression having already made its appearance, but hallucinations of vision are in no way necessarily contemporaneous with an attack of depression. Thus there would seem to be some additional factor that determines the appearance of hallucinations during the existence of this mental state.

Some patients volunteer the statement that the appearance of an hallucination is accompanied by a bad headache, and most complain that "the vision," or "the funny sounds," or "the filthy smells," always "make my head ache very badly."

Moreover, in no case where I have made a complete examination within twenty-four hours from the occurrence of an hallucination was scalp tenderness of the reflected visceral type absent.

Let me digress for a moment to recall to your memory how it comes about that diseases of the organs of the chest and abdomen may be accompanied by reflected pain in the head and tenderness of the scalp.[1]

All the thoracic organs and most of those in the abdomen receive fibres from the vagus. Impulses pass up these fibres to the head, and according to the usual rules that govern the appearance of reflected pain, should be reflected along the somatic branches corresponding to the visceral afferent fibres of the vagus. Now Gaskell suggested that part of the trigeminal or fifth nerve corresponded to the somatic branches of that set of nerves of which the vagus represents the visceral branches. And as a matter of fact pain impulses ascending the vagus from organs in the thorax and abdomen actually cause pain and superficial tenderness over certain portions of the scalp. But each area of pain and tenderness on the scalp does not stand in necessary relation to any one or more organs of the thorax and abdomen. We cannot say that one area is connected with the heart, another with the lungs, and so on. The relation is a segmental one, and each segmental area or group of areas on the trunk stands in relation with an analogous area or group of areas on the scalp. Thus it is immaterial what organ has been disturbed to produce, for instance, the seventh dorsal area on the trunk. So long as that area is present in a well-marked form there will be a tendency for tenderness to make its appearance over the temporal region of the scalp.

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[1] *Cf.* "On Disturbances of Sensation with especial Reference to the Pains of Visceral Disease." Part ii., Head and Neck. BRAIN, 1894, p. 436.

In this way the third and fourth cervical and all the areas from the first to the tenth dorsal are correlated with tenderness on the scalp.

Expressed broadly, this relation may be said to be that the higher the area on the chest the further forward its correlated area on the scalp. Thus the third and fourth cervical and upper dorsal areas will be associated with areas of tenderness over the nose and forehead; the seventh, eighth, and ninth dorsal with tender areas in the temporal, vertical and parietal regions; and the tenth dorsal with tenderness over the occipital region.

Thus affections of the organs in the thorax and abdomen will not only be accompanied by reflected pain over areas of the trunk, but also, if sufficiently intense, by headache and scalp tenderness.

Now the Gasserian ganglion must play a part in the production of this reflected pain and tenderness. It therefore seemed possible that so-called herpes zoster ophthalmicus, a disease accompanied by an irritative lesion of the ganglion, and associated with exquisite pain over the forehead, might also be accompanied by hallucinations. This turned out to be the fact, for out of eleven cases of herpes zoster ophthalmicus five had been the subjects of typical hallucinations of vision of the type I have already described. One saw a large white face at the bottom of the bed, one a "corpse," two saw figures standing beside the bed "misty, as if wrapped in a cloak," and one, even whilst walking the room at night on account of the pain, frequently saw a "shadow," without a face or limbs, standing in the room. In none of these cases was the cornea or iris affected by the inflammatory process. In no case had the patient ever experienced an hallucination of any kind on any previous occasion.[1]

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[1] *Cf.* Example 10, p. 429.

The presence of hallucinations of vision in these cases of herpes zoster ophthalmicus seems to show that the determining factor for the appearance of hallucinations is not the intensity of the general psychosis; for in these cases of herpes zoster the other elements of the psychosis, depression and suspicion, were but slightly marked, and under ordinary conditions, hallucinations being the last element of this psychosis to make their appearance, we should scarcely have expected them to be present. There must therefore have been some unusual factor in such cases. This I believe to be the fact that no pain was present elsewhere, except within the territory of the superior division of the trigeminal.

Thus it would seem that headache of the reflected visceral type is the only necessary concomitant to the appearance of hallucinations.

But in order that hallucinations may make their appearance the pain stimulus must have a certain bulk; it must have reached considerable intensity. If the pain suddenly makes its appearance in a person who has been for a time free from pain, this rapidity of appearance seems to more readily predispose to the appearance of hallucinations than if the pain had reached the same or even greater intensity more slowly.

If, at the same time, the resistance is lowered, as by fever or anæmia, an attack of headache seems to more readily conduce to the appearance of an hallucination than if neither fever nor anæmia had been present.

Not only must the headache be of sufficient intensity and the resistance lowered, but the appearance of hallucinations is very much aided by concomitant pain and tenderness on the trunk—for this pain and tenderness alone can be associated with depression, and the existence of this depression is a sign that the mental control has been broken. Thus many cases of chronic headache in which pain of the reflected visceral type is produced from other causes than disease of the organs of the thorax and abdomen may never suffer from hallucinations of any kind. But cases of disease of the organs of the thorax and abdomen that cause less severe and less continuous headache may be accompanied by hallucinations, owing probably to the way having been paved by the concomitant depression.

Thus headache of the reflected visceral type, particularly when accompanied by similar pain and tenderness on the trunk, is liable to be associated with hallucinations.

What determines whether the hallucination shall take the form of sight, sound, or smell?

It might be thought that the headache tended to be associated with what might be called a general hallucinatory state and that the form assumed by the hallucination was determined by the condition of some particular sense organ. An analysis of the cases where an hallucination occurred during the patient's stay in hospital shows that this is not the case. For in no case were hallucinations of sound or smell accompanied by any abnormality of either the ear or nose, nor was hearing or smell permanently affected. The only fault that was occasionally present in the eyes of those patients who had experienced an hallucination of vision was some slight error of refraction. This could in no way be responsible for the hallucination for it was absent in most of the cases.

Thus it is certain that no permanent error in the sense organ implicated was the determining cause of the form assumed by the hallucination.

It might be thought that the headache was accompanied by a general hallucinatory tendency, and that a patient who was once the victim of this psychosis would develop hallucinations of smell when brought face to face with food; hallucinations of hearing when the surroundings become free from noise; and hallucinations of vision in the dark. And there is little doubt that this view is in many ways correct.

But if thirty-four cases are put together where a patient experienced an hallucination whilst under observation in hospital, there were only four cases in which that patient, at any time, whether in or out of hospital, had experienced all three forms. In thirteen cases a second hallucination had been experienced in addition to the one observed in hospital, and in seventeen cases one form of hallucination only had ever been present.

But in a large number of these thirty-two cases the same form of hallucination had been repeated, not uncommonly, several times.

Thus, although the presence of headache of the reflected visceral type tends, under suitable conditions, to be accompanied by hallucinations, there seems to be some factor which determines the form that shall be assumed by the hallucination that appears.

Theoretically I was led to suppose that the form assumed by the hallucination depended on the position of the headache for the following reasons :—

We have, so far, spoken as if pain and tenderness over the scalp was only produced by disease of organs of the thorax and abdomen. But disease of such organs of the head as the eye and the ear will also cause pain and tenderness over the same regions. In fact, the whole forehead is peculiarly associated with the pain produced by diseases of the eye as are the vertical and parietal regions with painful diseases of the ear, more particularly such as are due to rise of tension from pent-up discharge. Thus, from a psychical aspect, there is a close relation between the forehead and the eye, the vertical and parietal regions of the scalp, and the ear.

Hence, on theoretical grounds, it appeared possible that, although these mental changes when present tended to be accompanied by hallucinations, the form assumed by the hallucination might depend on the position of the headache and its accompanying scalp tenderness.

The material at my disposal is too small to prove this theory, but a close examination of the sensory changes that accompanied the thirty-four hallucinations which occurred whilst the patient was under care in a hospital do not go against this view and in many cases tend to support it.

For out of nine visual hallucinations, superficial tenderness of the visceral reflected type was present over the forehead in every one, and in four the area of tenderness did not extend on the scalp further back than the forehead.

In twelve cases of hallucinations of hearing, tenderness was present over the vertical and parietal region in all, whilst in six it was confined to that territory of the scalp.

Moreover, support is given to this view by the fact that in the five cases of herpes zoster ophthalmicus, where hallucinations were present, in every case the hallucination was one of vision.

Thus, if we assume that when this psychosis is established the form assumed by the hallucination depends on the position of the headache or the area of the scalp tenderness, we can thereby explain the occurrence of hallucinations of vision and hearing. But those of smell still remain a difficulty; for in every case where an hallucination of smell occurred whilst the patient was under observation the headache was mainly in the temples, and superficial tenderness was present over the temporal regions on one or both sides. In six of these thirteen cases the tenderness was confined to the temporal region.

But I think the following considerations help us to understand the association between headache accompanied by superficial tenderness in the temporal region and hallucinations of smell.

These hallucinations are uniformly unpleasant. In no single instance have I ever been able to discover even traces of a pleasant hallucination of smell in sane persons suffering from visceral disease who were not delirious. Moreover, the patient always states that the smell "made him feel sick," "turned him off his food," or the like.

Now anyone who will examine his sensations when he is suffering from nausea, before vomiting has actually occurred, will recognise to what an extent the unpleasant aspect of the sense of smell is increased. Scents that it is true contain some unpleasant element, but which under normal circumstances are indifferent or even pleasant, become not only stronger but at the same time definitely repulsive. Everyone who has been sea-sick knows how offensive his friend's pipe becomes, and at the same time at how much greater a distance he can smell that pipe. And yet, perhaps, under normal circumstances there may be no smell that is pleasanter to him. The tarry smell of the ropes, and the smell of food are not only increasingly unpleasant, but seem to be actually stronger than before the feeling of sickness came on.

Thus it may be that there is a close association between the vomiting centre and that for smell; and it is easy to see the reason for such an association, for it is obviously protective, and serves to guard us against the ingestion of abnormal or improper food.

Now there is also a close association between the temporal area of the scalp and vomiting; for the temporal area seems to stand in relation mainly with the sixth and seventh dorsal areas on the body, the areas within which pain and tenderness make their appearance in most painful affections of the stomach. Moreover, when these areas of tenderness are present, from whatever cause, in considerable intensity, taking food is almost immediately followed by nausea and a desire to vomit.

Thus the temporal area of the scalp stands in relation to nausea and so indirectly to an exaltation of the offensive aspect of smells.

When, therefore, the psychosis I have described is once established and the mental field has become turned to visceral impressions, the presence of pain of the reflected type and tenderness over the temporal area, whatever its origin, will not only tend to excite nausea, but also, under favourable conditions, to produce an hallucination of smell.

In this way the presence of food by inducing nausea aids the development of such hallucinations of smell.

But it must not be supposed that the offensive smell of which the patient complains bears any direct relation to the scent of the food. For there is no element in the smell of cocoa and milk that corresponds to "a foul burning smell like very foul tobacco" any more than there is anything in the smell of roast meat that corresponds to fæces. Moreover, in most cases, the patients say that the smell is not confined to the food but is all about them, and one thought it was on his hands.

It is uncommon to find two hallucinations occurring together at the same moment. One patient told me she heard tapping at an upstairs window and, looking, saw a white face. But in the great majority of instances, if a patient suffers from hallucinations of more than one kind, these hallucinations occur on entirely separate occasions.

This bears out the contention that the determining factor in the form assumed by the hallucination is the local intensity of the headache and scalp tenderness.

Widespread scalp tenderness unaccompanied by marked local headache is unlikely to be associated with the occurrence of an hallucination. But not uncommonly the patient has long suffered from widespread scalp tenderness which, at the time of the hallucination, is reinforced by a rapid and vehement outbreak of headache over some particular area of the scalp.

Thus a patient with a progressive disease such as tubercular phthisis will tend to suffer from manifold hallucinations, for the area of pain and tenderness on the trunk shifts with the progress or varying incidence of the disease. At the same time the part of scalp affected and the greatest incidence of the headache will also shift and thus, when once the psychosis has been established, hallucinations of different kinds will make their appearance during the course of the disease in the same patient in association with the shifting incidence of headache and scalp tenderness.

CHAPTER III.—CHANGES IN ATTENTION AND MEMORY.

So far I have made no mention of the condition of memory and attention in those who suffer from visceral disease. This is because the causes that underlie the changes that occur are more complex and more difficult to unravel than those which underlie the symptoms of which I have already treated. It is impossible to place a series of cases that suffered from reflected pain into the one group and those that had no pain into the other group and to show that attention and memory were altered in the one and not altered in the other.

Many patients recognise the profound loss of attention that may accompany disease of the heart and lungs. First they notice that they cannot keep their attention on a book or newspaper even though they may be extremely fond of reading. They can no longer play chess or draughts and lose all interest in cards. Women find they cannot sit and sew as they were wont to do, though they never lose the power of knitting, which appears to be a purely mechanical process.

Memory for the remote past is not affected except in those extreme cases where the patient becomes slightly demented as, for instance, in some extreme and long continued cases of pulmonary tuberculosis. Patients who have suffered from some disease since childhood that has persistently impaired nutrition will tell you they have always had a bad memory. Some cases of congenital heart disease or of adherent pericardium are unable to remember when or where they went to school. Here, however, we have not to deal with the destruction of a memory of remote events but rather of an absence of memory for recent events that has persisted throughout the greater part of the patient's life.

If the disease comes on in adult life, when a memory already exists well stored with records of past events, the remote past is left untouched. Patients remember where they went to school, the character of their teachers, where they first went to work, and other incidents of remote memory.

Any diminution that takes place lies within the field of recent memory, and more particularly within that portion of it that is covered by their illness.

If a man has a special facility based on some particular development of memory, this aptitude is the first to be weakened. Thus one of my patients[1] was a porter at a tea warehouse. He prided himself on being able to send a parcel to any part of the United Kingdom without applying to a time-table or address book. This faculty became so well known that his friends used to search out difficult questions to put to him. Twelve months before I first saw him he developed pulmonary tuberculosis and began to waste, losing in all two stone. He also suffered from reflected pain and headache with each of the bronchitic attacks that signalised the advance of his disease. When I saw him there were signs of consolidation at the left apex, with early invasion of the lower lobe of the left lung. During a stay of six weeks in hospital his temperature was never over 99·4° F. For twelve months his memory had become very bad; for when told to send goods to any place at a distance he was obliged to look it up in a book. This has caused him great annoyance, for it was of this faculty that he was particularly proud.

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[1] Case 70, table 8.

Another male patient[1] showed exactly similar changes with the onset of tuberculosis of the lungs. He was 30 years of age, and for a year had suffered from a cough with occasional hæmoptysis. He had lost weight considerably, and lost two stone in the month that preceded his admission. His temperature was never above 99·4° F. during his stay in hospital. There were signs of consolidation at both apices, extending into the upper part of the lower lobe on the left side. He suffered from a good deal of reflected pain accompanied by superficial tenderness.

He was an upholsterer by trade, but at the time of his illness was also a betting man. When his illness, that he took to be "just a common cold," first came on he began to be troubled by a curious loss of memory. He could not remember the names of the horses or the odds in any particular race, and forgot how any particular horse had run in previous races. He repeatedly overlaid his book, could not remember his slips, and actually paid out money where it was not due. So bad did this loss of memory become that he was obliged to give up his profession and stick to his trade as an upholsterer.

But in addition to such special aptitudes memory is frequently diminished in other directions. Told to do a thing, the patient either entirely forgets or comes back to ask what it is he is to do. A waitress with mitral stenosis could not carry more than one order in her head at a time.

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[1] Case 65, table 3.

Closely allied to this is the difficulty in remembering an intention. Women go shopping and find they cannot remember anything they went for. They put things away and have no idea where they put them.

A deeper level of disturbance is shown when the patient forgets things he has seen or heard. A patient who could come home from a concert or musical play, and reproduce the airs to which he had listened, found that when his illness began he could not remember either what he had seen or heard. Many as they walk along the street see things they intend to tell, but when they reach home have forgotten what it was they saw.

A large number of patients with cardiac or pulmonary disease forget what they read; but this seems more directly associated with lack of attention than any of the other memory changes. They can read a column of the newspaper, or a few pages of a book again and again, and have no memory of what it contains. I have seen a patient with an aneurism read over with the greatest interest a long account of the murder that provided the excitement of the moment. But the same evening he was quite unable to tell another patient anything about what he had read.

The loss of due appreciation of time and of locality that forms so prominent a feature in cases of so-called confusional insanity is never present unless there has been marked alcoholic excess in addition to the visceral disease. The patients I have studied for this work always knew the day of the week, the month and the year. Taken round the ward or round the garden, they could find their way back again without difficulty. It is true that a few patients with cardiac or pulmonary disease cannot remember the way to places they knew quite well. But this appears to be a true loss of memory, and in no way resembles that loss of orientation so characteristic of so-called confusional insanity. Taken to the same place they could find their way back, but they fail when attempting to recall the way by which they went and returned.

These changes in memory and attention cannot be associated, like the other mental symptoms I have described, with reflected pain only. For although a considerable proportion of the cases in which reflected visceral pain was a marked feature showed more or less profound loss of attention and memory, there were also many cases without such pain that showed similar changes. [*Cf.* Tables 1 with 2 and 3 with 4.]

However, in those cases of cardiac disease where reflected pain, wasting and fever were absent, attention and memory were unaffected.

But if pain is not the only cause neither can continued fever be associated solely with this loss of memory and attention, for many of the cardiac cases in which it was most marked were afebrile.

Nor can wasting account for all cases, for many of those cardiac cases which show most profound loss of memory and attention show no wasting.

And yet wherever reflected pain is a marked feature of the disease, wherever fever is prolonged or wasting acute, then these changes in memory and attention will be marked.

Attention is the direction of thought at any moment to this or that special object in preference to any other. In order, then, that the power of attention should be unimpaired, it is necessary that the field of consciousness should not be dominated by any one presentation. Whatever the origin and nature may be of a presentation that dominates consciousness, the existence of such a state produces diminished power of attention. The mind is no longer free to react to each successive sensation or idea—its interrogative aspect is gone.

Thus the insane patient, who believes he is about to be hanged, pays little attention to what is going on around him. In the same way a sane person who is suffering from the pain of herpes zoster ophthalmicus will be unable to give attention to his work or to reading.

But these pain sensations do not cause a diminution in the power of attention only by the fact that they enter into consciousness as pain impressions. For in many cases where reflected pain is associated with marked loss of attention, consciousness is dominated not by the pain as such, but by the changes in feeling-tone with which that pain is associated. The patient does not complain of the pain, but rather of the depression that has come on since his illness.

It is in this way that we can account for those cases where attention has failed since the development of reflected pain, but where fever, wasting, or anæmia are absent.

On the other hand there is no doubt that where fever is prolonged, wasting, a prominent feature, or anæmia well marked, attention will fail simply from the rapid fatigue that occurs where the vitality of the nervous system is lowered.

Now, as I have already pointed out, memory may be lost in consequence of diminished attention. A patient will not remember what he has seen or heard, or where he has put things, simply because during that time his attention has not been fixed on what he has seen or heard, or on what he is doing. Thus the domination of consciousness by some continuous presentation or fatigue, due to lowered vitality of the nervous system, will produce loss of memory for events that occurred during the period of the illness by causing loss of attention. Images that should have been stored up are not registered, and memory of this period does not exist.

But in visceral disease it is not only from lack of attention that memory is diminished, for memory is not only lost for the actual period of the illness, but also for a time preceding its onset, in which memory images have been certainly stored in the normal way.

The first cause of this actual loss of already formed memory images is the lowered vitality of the nervous system, produced by continuous fever or profound wasting.

I take as an instance of such loss a very intelligent man of thirty-six, an insurance agent, who died from ulcerative endocarditis.[1] Four months before his admission to the hospital he became short of breath. He then began to have shivering attacks in which he became very hot and then very cold. He did not think he had wasted, and on admission showed no signs of emaciation. His temperature reached 102° every night, usually falling to normal in the morning. He had never had any pain of any kind. A double aortic and mitral systolic murmurs were well heard, and the latter was conducted to the back.

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[1] Case 31, table 2.

This man was in no way depressed and showed no symptoms of the phenomena I have brought before you in these lectures.

His attention was but little impaired and although physically so ill he could read a book through without difficulty. But at the end he knew very little about what occurred at the beginning of the book and a very few days after he had finished it he did not even know what was in the last chapter. Although he could remember his childhood he had forgotten much that preceded the onset of his illness. He forgot what he saw in the street before he reached home. He was easily tired by any attempt at recollection.

This seems to have been a case where memory was lost owing to the diminished vitality of the nervous system produced by continued fever.

But when consciousness is dominated by images connected with the system of psychical dispositions that stand in relation to visceral reflected pain, memory will be weakened apart from the diminished freedom of attention so produced. For a system then becomes elevated into the central field of consciousness that has connections and dispositions widely different from those of the content of normal consciousness. Thus it will no longer be possible for a patient whose consciousness is dominated by a system of visceral origin to recall outlying images or dispositions that belong to his normal field. His memory will therefore be profoundly diminished, not because he thinks about his pain, but because this pain has changed the system that occupies the central field of consciousness.

Then again we must remember that states in which feeling-tone or emotion occupy consciousness are extremely poor in memory images. Most of us can recall the look of the spot where we have been profoundly miserable and have a clear picture of all the surrounding circumstances, but few of us can reproduce the state of misery we experienced on that unhappy occasion. In the same way few can reproduce the sensation of a toothache although every object in the dentist's room can still be pictured as clearly as on the day the tooth was drawn.[1]

Thus not only is consciousness in these cases of visceral disease dominated by a system of presentations and predispositions which preclude the appearance of the usual content of memory, but this very system, consisting as it does mainly of states of feeling-tone, and emotion, is in itself for most of us but little capable of representation. Memory, therefore, is the poorer at the moment, and is not enriched by this state when it has passed away.

Thus memory tends to be irrevocably lost during the acute stage of the illness, because no images are laid down for future representation.

At the same time the memory of that period antecedent to the illness in which the images are not firmly fixed is also diminished, owing to the domination of consciousness by a system of presentations and predispositions other than that which usually occupies the central field. As soon as the attack ceases and the patient again returns temporarily or permanently to the normal, this loss will be recovered. The system that has dominated consciousness during the existence of the visceral pain will cease to occupy the field, and with the return of the normal content to the centre of the field comes back its attendant connections and predispositions.

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[1] In the majority of the patients included in this research memory was essentially visual. This is also the case with most of my friends and with those patients of the richer and professional class with whom I have been brought in contact

But I have come across three cases of that interesting form of memory so accurately described by Ribot "Recherches sur la Mémoire Affective," (*Revue Philosophique*, tom. xxxviii.), and called by him "true or concrete affective memory." In this form of memory feeling-tone, moods, or emotion are represented directly, without the intermediation of visual auditory or tactile representations as in most of ourselves. In addition those gifted with this memory are capable of calling up scents in the same direct way that most of us can call up a picture of some familiar place, scene, or person.

This is an uncommon form, and no such case is included amongst the patients on whom this research is based.

CHAPTER IV.—CONCLUSION.

WE have now seen that the presence of pain of the reflected visceral type tends to be associated with a mental state, containing amongst other conditions, a sense of ill-being and a causeless suspicion.

In conclusion, let us attempt to discover, if possible, firstly, the way in which this association is brought about between pain and a peculiar mental state, and secondly, the causes that may have led to this association.

The lower we pass down the animal scale the more purely is the life of the individual determined by visceral impulses. On the other hand the aim of human development is to keep such impulses in the background in order that the mind may be ready to receive impressions of all kinds from an ever shifting environment. Under normal circumstances visceral life takes place outside consciousness. At the most, consciousness is stirred by some slight alteration in feeling-tone, so entirely unaccompanied by sensation within the field of consciousness that we remain ignorant of the place whence it comes, or the physical activities that give it birth. The visceral field is pushed out of consciousness, and its records remain only as latent dispositions.

But a complete change takes place when reflected pains of visceral origin come into existence. These sensations produced by abnormal activity of the viscera crowd into consciousness, usurping the central field of attention. Now when pain of somatic origin, as for instance that of an injured limb or a burn, enters consciousness, it occupies attention, but brings nothing in its train. We grow miserable with the pain of the injury, with the restlessness and inability to sleep, but our attention is occupied only by the desire to place the injured part in the least painful position. Our sleep may be troubled with dreams, but they are either dreams of pain such as that a cat is clawing the injured part, or worrying dreams about work and the affairs of the day. We do not wake frightened or in tears. We are not subject to sudden unaccountable attacks of low spirits. Nor are we suspicious of those around us. If consciousness is disturbed it will be by pain, and pain only.

On the other hand reflected pain of visceral origin brings in its train all those images and dispositions which exist normally at the fringe of or entirely outside the field of consciousness. All those sensations that are associated with visceral activity, which do not exist in consciousness under normal conditions, come to the surface. The patient's character appears to be altered, for the content of his consciousness is changed. He will become moody, at one time unduly exalted, at another depressed without cause. Reason is displaced and he is the victim of each passing wave of feeling-tone, and he will have lost control over the expression of his emotions and of his temper.

That barrier which the normal mind sets between conscious life and that of the viscera, the integrity of which depends on a high potential of vitality in the nervous system, has been broken down.

Now, in the life of a woman this barrier is less stoutly fixed than in that of a man, owing to the periodic occurrence of menstruation and its attendant mental manifestations. The resistance to be overcome in a woman is therefore less than that in a man. This accounts for the greater ease with which all visceral manifestations can be produced in women compared with men.

But in addition to a great susceptibility to visceral activity reflected pain brings in its train a series of anti-social mental states. The patient is impelled to separate himself from his fellows by an impulse to be alone, a sense of some unknown impending ill and suspicion of those around him. Everything urges him to get away by himself. He is permitted no choice of "staying and seeing it out," like those who are suffering from a true delusion of persecution, for reason plays no part in the matter, and if the psychosis once gains the upper hand he is delivered over to a blind impulse to shun his fellows.

But man is a gregarious animal, and civilised man is peculiarly characterised by his tenderness to those who are ill. Moreover, in the intervals when he is not dominated by these mental changes the patient recognises that far from those around him being against him they are, if anything more solicitous of his welfare than before he was ill. His fellow-workmen or his master make his job easier for him; his friends help him with his home or children; his wife is more affectionate than before.

Thus the effect produced upon his actions by the mental state induced by his disease serves directly, in a civilised community, to make his lot harder than it would otherwise be.

How then is it possible to explain the origin of so pernicious an association as that between visceral pain and these anti-social impulses?

At the close of these lectures perhaps I may be permitted to indulge in fantastic speculation. The oldest system in the body is that to which the name autonomic has recently been given. Under this name we understand not only all that is known to the anatomist as the sympathetic system, but also all those parts in the central nervous system which stand in connection with the viscera. It still retains traces of a primitive segmentation lost elsewhere. The sensory supply of the heart is still arranged as if that organ were a tube with the ventricles situated headward to the auricles. The plan on which this nervous system is laid down is related to that of invertebrates more closely than to the cortical system of man. It is essentially the system of animal, as opposed to human activity.

Now, when an animal is wounded or ill, he is killed by his fellows without mercy. A wounded animal therefore crawls away either to recover or to die in his hiding place. But an instinct that is salutary for an animal or for the lowest savage is entirely opposed to the highest development of the human mind. It is therefore no longer associated with those parts over which the highest centres of man have the completest control, but still remains attached to sensations from the visceral system.

Hence, when the mental states that have occupied us in these lectures appear in consciousness they seem an intrusion from without, an inexplicable obsession that can neither be controlled nor subjected to logical analysis.

All such states have since the earliest times been classified under the name hysteria, and I shall be satisfied if in these lectures I have been successful in relating to their proper position a small band of symptoms included under that vast and heterogeneous group.

APPENDIX.—CASES REPORTED IN FULL AS EXAMPLES OF THE MENTAL CHANGES DESCRIBED ABOVE.

Example 1. [Case 23, table 1.] *To illustrate the hallucinations that may appear with cardiac disease.*

Edward B., aged 42, has been under my care at the London Hospital from August, 1900, until the present time. On February 27, 1901, he was admitted under Dr. Percy Kidd, and discharged March 8.

The following account embodies the results of many examinations at different times during the last twelve months.

§ 1. *History.*—He has had two attacks of acute rheumatism, one at 16 and one at 21.

For fifteen years he has known that his heart was affected, but he dates his illness from 1898. Before this date he had suffered from pain occasionally; but since then pain and headache have been almost continuous.

He is a teetotaller and non-smoker, is married and has nine children living and healthy.

§ 2. *Physical condition.*—He is a well-built, intelligent man of 42, a carpenter by trade. He shows no signs of anæmia or cyanosis. He is spare, but there is no general wasting.

He complains of pain in the left side and down the left arm on the ulnar aspect. This pain is always accompanied by more or less superficial tenderness over an area, on the left half of the chest only, extending from the second space above to the level of the ensiform below and down the ulnar side of the left arm to the elbow. (Dorsal 2 to D. 7.) Occasionally there is also slight tenderness over the supraclavicular fossa and back of the neck on the left side. (Cervical 3 and 4.)

He also complains much of headache over the left side of the head, accompanied by scalp tenderness mainly over the forehead, but also extending back to the temporal and vertical regions. The right half of the head has always been entirely free from headache and scalp tenderness.

He is short of breath on exertion, but is able to do light carpentering unless the pain becomes troublesome. He then rests and can return to work in a few days.

The pulse is usually about 74, with a small stroke and collapsing character.

The apex beat is situated in the sixth space, 4¾ inch from the middle of the sternum (1 inch external to the nipple line). Cardiac dulness begins above at the third space, extends 5 inches to the left of the middle line and to the right edge of the sternum. A typical double aortic murmur can be heard over the base of the heart, the systolic conducted into the neck, the diastolic down the sternum. At the apex a systolic murmur is heard conducted to the midaxilla only. The first sound at the apex is short and very sharp, is loudly heard in the axilla and at the angle of the scapula. The second sound is usually absent from the apex.

The lungs are unaffected.

The liver is not enlarged and there has never been fluid in the abdomen.

The tongue is clean, bowels regular, and he never suffered from nausea or vomiting.

The knee-jerks are normal and there are no abnormal signs in the nervous system.

The urine contains no albumen or sugar.

§ 3. He is very intelligent and gives an extremely good account of his symptoms.

He frequently wakes at night to see a figure standing at the bottom of his bed on the left side. This figure is either white or grey. He can see the face but cannot accurately discern its features. It resembles a face and figure seen through a gauze. It is never "dressed like ordinary people but seems to be draped in some pale thing." This figure always seems to be looking at him. It lasts for many minutes and disappears without moving away. It seems to stoop down and to become hidden at the foot of the bed. Many a time he has got out of bed to look for it or has waked his wife and asked her to look at the figure standing at the bottom of the bed. When it first began to appear he used to be very frightened, his heart beat rapidly, he shivered and sweated. He can lie and look at the figure without being frightened of it, but he still shivers and sweats every time it appears. He says, "I don't seem to be afraid in my mind any longer, but I am still just as if I was afraid."

On August 19, 1900, I saw him on the morning following an hallucination of vision of this kind; at that time he was having hallucinations almost every night. He was lying awake with a very bad headache "at the back of the eyes," when the figure appeared to the left-hand side of the foot of his bed.

When I saw him next morning the left half of the scalp was tender over the left half of the nose, the forehead, temples and vertex. The right side of the head was free from pain or scalp tenderness.

On the body the tenderness occupied the cervical 3 and 4, dorsal 2, 3, 4, 5, 6, 7 and 8 areas, on the left side only.

Vision was $\frac{6}{6}$ in both eyes, there was no error of refraction. No changes were present in the fundus oculi except that the vessels are seen to pulsate (aortic disease).

When in the hospital in March 1901, under Dr. Kidd's care, he twice had a typical hallucination of vision, and exactly similar scalp tenderness was observed and noted by Dr. Salaman.

§ 4. He constantly wakes at night to hear a loud ticking in the wall to the left of his bed. It lasts for several minutes after he wakes. At first it frightened him, and he would get out of bed and tap the wall to see if he could stop it. His wife has never been able to hear this sound; sometimes he will hear it every night, sometimes it will go away for a month or more.

He had no auditory hallucinations when in hospital in March 1901, under Dr. Kidd.

His hearing is good, and the ears are absolutely normal.

§ 5. He has never had any hallucinations of smell or taste.

§ 6. Whenever his pain is troublesome he becomes liable to attacks of low spirits, and I have seen him in these attacks on several occasions. They come on quite suddenly, and he has an intense desire to cry. He goes away by himself and "has it out." He does not want to be spoken to, and if his wife, or anyone in the ward, spoke kindly to him when in this condition, the tears would come into his eyes. He had a feeling as if some unknown ill was about to befall him, and on one occasion I found he had written his name and address on a piece of paper and pinned it inside his coat, before coming up to the hospital. When in bed in the hospital he had several attacks in which he hid his head under the bed clothes and cried.

Music is very disagreeable to him when he is in this state. He says that when at home, if the piano is played next door, it causes him to be downhearted and he lies in bed and weeps.

He has a strong visual memory, and as one of the attacks was clearing away he made the following observation (June 17, 1901): "When I try to think of my home, the room looks all dark. I can't picture the cloth on the table, and yet I know there is a light one with a picture of the Transvaal War. There are bright mottoes all round the room, and the room, I know, is an uncommonly bright one, for my wife loves bright things. But somehow, I don't seem able to picture the room as I know it is, but all dark and miserable looking."

§ 7. He frequently has a feeling as if he was very strong. "Sometimes I feel as if I were 80, sometimes as if I were 18." He then wants to lift something, he grips his plane and starts, a heavy piece of work. But the exertion brings on palpitation and pain, and he is obliged to sit down.

The depression and exaltation alternate, but he is very definite in his statement that the exaltation comes on when the pain goes away, and that, with him, it follows the depression in most cases.

§ 8. His memory has become bad lately, particularly for things that have happened recently. This has become particularly obvious in his work. He works for a railway and has jobs belonging to many different stations and shops on the line. When the work is finished he cannot now remember for which place it is intended. Up till eighteen months ago he never had any difficulty in remembering his work.

He forgets what he reads, what he sees in a theatre, and even the titles of songs he has heard at a concert.

Remote memory remains excellent.

§ 9. He now finds great difficulty in "thinking a thing out." He cannot "take out the quantities" of material required for a job in his head as he used to do.

He can no longer keep his attention on a book or a paper as he used to be able to do; for he was a great reader and for years he had books from the free library. But for the last six months he has not taken out a single book.

§ 10. He constantly has a feeling that his fellow workmen are against him, and when in hospital felt sure the nurses thought he was not ill enough to be looked after.

When this feeling first started he told the people in the workshop that they thought him lazy. He was perfectly satisfied when they said it was nonsense. Now he knows these feelings are "all fancy," and has been able to control himself sufficiently never to charge anyone with being against him.

Example 2. [Case 15, table 1.] *To illustrate the mental changes that may accompany disease of the mitral valves.*

Alice T., aged 27, was admitted to the London Hospital, under my care, May 29, and discharged July 11, 1901. She is still under my care as an out-patient.

§ 1. *History.*—At the age of 12 she had suffered from chorea. For two or three years before admission the knees and shoulders had been swollen on several occasions, but she had never kept her bed. She had had many attacks of tonsilitis.

Up to the age of 21 she had worked as a shop assistant, but then had to cease work on account of cough and shortness of breath.

For two years before admission she had had pain in the left side on many occasions.

Her legs had begun to swell occasionally for some months before admission.

She had not menstruated for twelve months before admission. It had always been irregular, but painless.

§ 2. *Physical condition on admission.*—She was a light-haired, well-built, intelligent woman of 27. She had been a shop-assistant. There were no signs of anæmia, but there was a dark red flush on either cheek, and the red of her lips had a slight bluish tinge. She was not wasted.

She complained of pain in the chest, mainly between a line drawn round the body at the level of the nipples, and the umbilical line; greater on the left side than the right. This pain was accompanied by superficial tenderness over the fifth to the ninth dorsal areas, and also to a less extent over the third and fourth dorsal, and third and fourth cervical. This tenderness was well marked on the left side, but was patchy and of slight intensity on the right half of the chest.

She also complained of headache, mainly in the temples, but also on the vertex. This pain was accompanied by tenderness over the forehead, temples, and vertex on both sides, the left more marked than the right.

She was short of breath on the least exertion, and could not sleep unless she were propped up with several pillows.

The legs were not swollen.

The pulse was 76, regular, with a small stroke and maintained tension.

The apex beat was situated in the fifth space 4 inches from the middle of the sternum (half-inch external to the nipple line), and no thrill was felt. Cardiac dulness began above at the third space, extended 4½ inches to the left of the middle line and to the right border of the sternum. A systolic murmur was heard at the apex, and was conducted to the right as far as the epigastrium. To the left this murmur was not heard further than the midaxilla. The first sound at the apex was short, but sharp and loud, exactly resembling the typical first sound in mitral stenosis. The first sound was well heard at the angle of the left scapula. At the apex the second sound was reduplicated, but over the pulmonary area was accentuated only. No other murmurs were heard.

She suffered from no cough, but many dry râles were present over the base of the left lung.

The liver was not enlarged, and there were no signs pointing to fluid in the abdomen.

The tongue was clean and moist; the bowels tended to be confined. There was no vomiting.

The knee-jerks were normal, and there were no signs of any disease of the nervous system.

The urine was acid, specific gravity 1020, contained a trace of albumen but no sugar.

§ 3. She is a quiet, intelligent woman, and always gives a remarkably sober account of her symptoms.

During the period preceding her admission to the hospital, when she was an out-patient under my care, she frequently complained of smelling horrible smells. Sometimes it was as if something was burning, sometimes it resembled "fish that had gone a little tainted." For instance, just before admission, whilst at dinner, she smelt a horrible rotten smell that turned her entirely from all food. No one else could smell it.

When in the hospital, on June 1, 1901, at 9 p.m., she suddenly smelt a tainted smell, as if something had gone bad—"like meat that had gone nasty." It made her feel sick, but she did not vomit. She was in bed at the time, and there was no food of any kind in her neighbourhood. She had not been asleep. She called the nurse, who was unable to smell anything. Then she knew it was one of her "fancies" to which she was well accustomed.

I had seen her half-an-hour before this occurrence and had made a complete physical examination. The physical signs were those given above. Her sense of smell was good, and no abnormality was seen in the nose. Taste was excellent, and the tongue was clean and moist.

The next morning (June 2) she told me of what had happened after I left her, and I again made a complete examination of the nose, mouth, tongue, and of the condition of the chest. She was still suffering from temporal headache and marked superficial tenderness was present over the forehead, temples, and vertex on both sides.

On several subsequent occasions during her stay in hospital she experienced a similar "rotten" or "tainted" smell; but in every case, excepting in the instance detailed above, the hallucinations occurred during the daytime.

§ 4. On admission she complained that during the last two years, and especially just before admission, she had heard sounds that no one else could ever hear. Sometimes these sounds took the form of a loud knocking in the next room or in the street—"as if in the night somebody was doing a terrible lot of work either inside or outside the house." She has also heard bells ringing at night—"sometimes as if they were outside or at a distance, sometimes as if they were closer, in the room." Her mother could never hear these bells or the other sounds of which she has complained. The knocking sounds were always unpleasant and frightened her. The bells were sometimes pleasant, but sometimes worried her "on account of their horrible jangling."

On admission her hearing was excellent. A watch was heard at 18 inches from right and from left ears. The tuning fork on the forehead showed normal conduction. Examination of the ears revealed no wax and the membrane on both sides was normal and showed no cupping.

During her stay in hospital she had no hallucinations of hearing.

§ 5. During the two years preceding her admission to hospital she has occasionally woke at night to see a "darkish thing" come in at the door of her room. "It is a man, and yet it does not seem to be a man." "It seems too large for a man, and its face is all dark." "Its clothes don't seem as if they were made like ordinary clothes, but seem to have grown to it, and that is one reason why it does not seem like a man." It always frightens her and makes her "creep all over," and sweat profusely. Occasionally she had seen this figure in the corner of her room when she entered it at night. It made her stand still with fright.

On admission her vision was R. $\frac{6}{6}$ well, L. $\frac{6}{6}$ poorly, Jäger 1 R. and L. The fundi were normal, and there was no marked error of refraction.

She experienced no hallucination of vision during her stay in hospital.

§ 6. She complained of frequent attacks of low-spirits, and on July 7, 1901, I saw her in a typical attack which came on when she got up in the afternoon. She went out of the ward into the bathroom and cried. Why she cried she did not know, but she had a feeling as if something was going to happen. Whilst in this condition she thought about her home, and the only picture she could call up was "as if it was all muddled and upside down; things were not hanging straight, and all the furniture was in disorder." Next day she told me she had determined to discharge herself because she wanted to go home "to see if it was really like this."

She had several similar attacks during her stay in hospital, but they became less frequent towards the end of the time.

§ 7. She has frequently had a feeling as if she wants to get up, "rush about and do a lot of work." "I feel more than right—as if I had got extra strength." Thus, one day when in the garden of the hospital, she felt she could race up the steps to the ward, as if she had nothing the matter with her. She started to try but had to sit down.

This feeling generally came on after an attack of low spirits, or if she woke in the morning with little or no pain. Under such circumstances she wanted to help the nurse clean the ward and lift the beds about.

§ 8. Her memory had been failing for some little time before her admission to hospital. She could not remember where she had put things, what she had intended to do, or what she was told. She had had a strong visual memory, and even during her stay in hospital could picture fairly well; but she volunteered that this power, which was one of her greatest pleasures, had become much less in the few months preceding her admission.

In hospital she could not start any song by herself, whereas before she had had "a quick ear," and was fond of singing.

She was a great reader, and read several books through during her stay in hospital. Yet she could not remember the plot of the last book she read, or the name of the book standing beside her bed (July 9). She could not keep her attention on things as she used to do.

Memory for remote events up till about two years ago was good.

§ 9. Before admission to the hospital she had frequently had a feeling that people were talking about her when she knew it was not true. She had accused her mother, brother, and sister of being against her and of wanting to get rid of her. On the afternoon of June 1 (when in hospital) she told the next patient that the nurses and sister were going to get her turned out of the hospital; that "they just give me some medicine to please me but think there is nothing really the matter"; "they all want to get rid of me." All the time she knew it was untrue and shortly afterwards told me that she had made these statements but was certain they were untrue. She said, "I feel I want to make a complaint, and it is very hard to keep it back although I know it is not true," and "for a time I am quite satisfied if they tell me it is nothing."

§ 10. She very much improved during her stay in hospital. The pain became less severe and the attacks of depression less frequent. She was much less short of breath and was able to be up all day and to work about the ward without discomfort. The physical signs remained unaltered.

Example 3. [Case 17, table 1.] *A case of disease of the mitral valves. To illustrate more particularly the hallucinations of hearing that may accompany visceral disease.*

Emily E., aged 36, a widow, was admitted to V.P.H., under the care of Dr. Sainsbury, on November 25, 1893, and discharged on January 9, 1894.

§ 1. *History.*—She had had an attack of rheumatic fever at the age of 13, and at the age of 27 another attack which left her with shortness of breath, palpitation and pain in the left side.

In 1886 she had suffered from slight "inflammation of the lungs," and in 1889 suddenly spat blood. She had been liable to blood spitting from that time until her admission to the hospital.

She had been liable to "quinsies" ever since she was a girl.

Menstruation began at 17, and usually ceased whenever her general condition became worse. Pain usually came on with the onset of the flow, particularly in the back and thighs.

She was married at 17, and had had two children who died apparently of tuberculosis. Her husband died of phthisis.

There was a strong history of phthisis on both sides of her family.

§ 2. *Physical condition.*—She was a medium-sized woman of 36, with a sallow face and slightly dusky complexion. Her lips were a little pale. The ends of the fingers and toes were cyanosed, but the lips and ears were not blue. The ankles had been swollen but were not œdematous on admission. She was somewhat wasted, and said she had been getting thinner for two months.

She complained of pain in the left side below the heart under the angle of the left scapula. This pain was accompanied by marked superficial tenderness, over an area of the left half of the chest only, bounded roughly above by a line round the body at the level of the third rib, and below by a line at the level of the ensiform cartilage [dorsal 4, 5, 6 and 7.][1]

She also complains greatly of temporal and vertical headache on the left side. This was accompanied by scalp tenderness over the orbit, temple and vertex of the left side only.[1]

She was short of breath on exertion, and was a good deal troubled by cough. She could not lie flat in bed at night.

The pulse was 100, regular, and with a small stroke and maintained tension.

The apex beat was situated in the fifth space just internal to the nipple line. No thrill was felt. Cardiac dulness began above at the third space, extended to the left to a point just internal to the nipple line and to the middle of the sternum on the right. A systolic murmur was heard over the apex of the heart which was not conducted to the angle of the scapula. The first sound at the apex was short, sharp, and very loud. The pulmonary second sound was accentuated.

Respiration was hurried and both costal and diaphragmatic. There was no dulness over any part of the lungs. The breath sounds were universally feeble, vocal resonance poor, but no râles were audible anywhere.

The tongue was furred, with red edges. Appetite was poor. She did not vomit during her stay in the hospital. The bowels were confined.

The liver was not enlarged, and there were no signs of fluid in the abdomen.

The knee-jerks were normal, and there were no signs pointing to disease of the nervous system.

§ 3. She said that for the last two years she had suffered from sounds that no one else could hear, and had become quite used to hearing things that were not there. Thus, although she lived at the top of the house she frequently heard tapping at the window of her room.

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[1] I have given the actual distribution of the tenderness on the body and scalp that accompanied the hallucination of hearing on December 4. During her stay in hospital the tenderness was sometimes more, sometimes less extensive, but always tended to lie over the area from D4 to D7, on the left side and over the temple and vertex on the left half of the scalp.

On December 4, she heard a tapping on the wall close to her bed in the ward. She was startled and "upset," but not actually frightened. This lasted half an hour, and I was able to assure myself that there was no external cause for this sound. She was then suffering from headache mainly over the top of the head on the left side, accompanied by the tenderness described above in § 2. Her hearing was good, and examination revealed no abnormality in her ears.

This was the only hallucination of hearing during her stay in hospital.

§ 4. Before she came into the hospital she had occasionally smelt foul smells no one else could smell. During her stay she several times smelt rancid oil burning in the ward, which the other patients could not smell. On the morning of December 8 the cocoa seemed smoky and burnt; no one else could smell it. She was suffering from her usual temporal and vertical headache.

§ 5. Before admission she had seen a white figure in "long loose robes" standing by her bed. It seemed to be about a yard from the bed, and was very pale. It always seemed to advance towards the left side of her bed, and once she had put out her hand to push it away. She had no visual hallucination during her stay in hospital.

§ 6. For more than a year before she came into the hospital she had been subject to attacks of causeless depression. She felt she was going to hear bad news, wanted to go away and cry, and could speak to nobody. She had at least one attack during her stay in hospital.

§ 7. Her memory had been bad for three years. She would forget what she had seen, where she had been, what she went out for, and where she had put things.

Her memory for the remote past was poor, but for recent events was exceedingly bad.

§ 8. She was fond of reading, but could not keep her attention on a book, and forgot what she read.

§ 9. She had no feeling that people were against her whilst she was in hospital.

§ 10. She became on the whole freer from pain as long as she was kept in bed, but the pain returned when she got up. The physical signs remained unaltered, and she left the hospital slightly better than when she came in.

Example 4. [Case 12, table 1.] *A case of aortic aneurism; to illustrate the alteration in mental state that may be associated with the development of visceral pain and the return to normal that may accompany the disappearance of such pain.*

Jane R., aged 53, widow. Admitted to Marylebone Infirmary, November 17, 1895, and last seen by me April 15, 1896 (Mr. Lunn).

§ 1. *History of present illness.*—In October, 1894, she noticed a pain in the interscapular region, which seemed to settle at one place in her back. On January 24, 1895, she went to the Brompton Hospital and was immediately admitted. Thence she was transferred to the Middlesex Hospital, and from there to the Infirmary. She had been in bed during most of the time she was in hospital, and on admission to the Infirmary the pain was much less severe than it had been. But if, when allowed up, she walked fast her breath became short.

She had never been pregnant. Menstruation ceased twelve years before admission. She had never had any illness excepting "low fever," and had been very temperate.

§ 2. *State on admission.*—She was a well-built, bright, cheerful woman of 53. No signs of anæmia. No œdema of face, legs, or back. She showed no obvious wasting.

She was entirely free from pain and there was no marked superficial tenderness. She was not short of breath when in bed.

The heart's apex beat was difficult to localise. Cardiac dulness began above at the third space, extended to the left nipple line (3¼ inches from mid-sternum) and, on the right, to the middle of the sternum. Over the upper part of the sternum and the second right intercostal space a soft blowing systolic murmur was heard abolishing the first sound. Over this area the second sound was accentuated. Elsewhere the sounds of the heart were well heard and there was no murmur.

Very slight pulsation was visible above the manubrium sterni, and if the hand was firmly applied over the second right costal cartilage a distinct thud could be felt with each heart beat. No tracheal tugging was present.

The pulse was 68, with a rather small stroke and no regurgitant characteristics. The two pulses at the wrist were equal in time and force. Capillary pulsation was absent.

The left vocal cord did not move well on respiration.

Beyond slight signs of emphysema the lungs were normal.

The tongue was red and moist, heavily lined and furrowed. Appetite was poor. No vomiting. The bowels were confined and could only be opened with aperients. The urine was normal.

The left pupil did not dilate to shade but there was no enophthalmos or drooping of the left eyelid. With this exception there were no abnormal signs in the nervous system.

§ 3. She remained free from pain, bright and cheerful until about January 7. She then began to complain of pain in the left side and headache across the forehead. This culminated in a sudden attack on the afternoon of January 12, during which she screamed with pain. It was situated mainly over the epigastrium and back, but there was widespread tenderness over the areas from the second dorsal to the ninth dorsal, more marked on the left side but present also on the right. The headache was mainly frontal and temporal, and was accompanied by marked tenderness over the whole forehead and over the temples on both sides.

Except that perhaps the pulsation was somewhat more marked over the second right intercostal space, the physical signs remained unaltered.

Mentally, however, she had changed completely. She was sleeping poorly and had been troubled by horrible dreams. On January 11, in the middle of the night she woke and saw a black figure come into the ward. It was so tall that it could only just get in at the door. The face was black. It came forwards towards her bed from the left. She was much frightened. She had never before experienced an hallucination of vision.

On both January 12 and 13 she had an attack of acute depression in which she cried, wanted to be by herself, and had a feeling that something was going to happen.

On January 14 she said "this week I have begun to think that the people in the ward are talking about me, and saying I ought to get up and go about, for I am not really ill. But I believe it is all my fancy."

§ 4. On February 7 she was beginning to improve, and by the beginning of March was free from pain and headache and superficial tenderness was almost completely gone.

Her mental state again changed. She slept better and had no hallucinations. Her last attack of causeless depression occurred during the third week in February. The suspicion had entirely ceased, and she had become the sober cheery woman she was on her first admission.

Example 5. [Case 72, table 3.] *To show the mental changes that may be associated with progression of pulmonary tuberculosis towards a fatal issue.*

Bernard McD., aged 21, single, was admitted to V.P.H., under Dr. Harris, July 9, discharged October 10, 1894.

§ 1. *History.*—Whilst serving in the Marine Artillery he broke his ankle and was invalided home in May, 1892. For five months he nursed a friend who was dying of "consumption of the throat."

After the death of his friend he felt languid, became liable to "colds" and had an attack of "influenza."

But in August, 1893, he enlisted in the Cape Mounted Rifles. On the voyage out he again "caught cold" and lost 7lbs. He became steadily worse and in May, 1894, was invalided home with phthisis.

He denied syphilis and gonorrhœa.

There was a strong family history of phthisis. One of his brothers and one sister are affected, and one of his paternal uncles died of consumption.

§ 2. *On admission* he was a well-built man of 5ft. 10in., but wasted and cachectic. He had lost three stone in eighteen months.

The temperature was irregular, reaching 101° at night.

There was considerable dyspnœa. The cough was paroxysmal and the expectoration not infrequently tinged with blood.

He complained of pain, mainly over the neck and upper part of the chest on both sides. This was accompanied by much superficial tenderness.

Over the upper lobe on the right side, both in front and behind, the percussion note was diminished, the vocal resonance increased and the breath sounds weak but bronchial. Over this area râles were heard which behind involved the upper part of the lower lobe. The remainder of the chest was unaffected.

The circulatory system was normal and pulse 84.

The tongue was clean and red at the tip and the appetite was fairly good.

Neither nausea nor vomiting were present, and the bowels were regular.

§ 3. *Progress.*—By August 3, the disease had rapidly progressed down the right side so that the base of the lung was affected. By the end of August not only was the whole left side affected, from apex to base, but over the right apex the note was dull, the breathing was bronchial, vocal resonance increased and râles were audible. By the middle of September the disease had extended into the upper part of the lower lobe on the right side, and at the end of September râles were heard around the angle of the right scapula. On discharge (October 10) the right base had become permanently affected. He died three weeks after discharge.

Throughout the whole time his temperature was irregular, frequently reaching 102° at night, but always falling to normal in the morning.

He lost weight steadily for the first seven weeks, then gained slightly, finally losing again.

His pain and the extent of the superficial tenderness greatly increased, and came to lie more definitely over the sixth, seventh, eighth and ninth dorsal areas, first on the left and then on both sides. The maximum incidence of the headache and scalp tenderness, which at first lay over the forehead, shifted back to the temples and finally to the temples and vertex.

His tongue remained clean and red throughout but appetite varied greatly. He became subject to attacks of diarrhœa in which the stools were offensive. He had frequent attacks of nausea but little vomiting.

§ 4. Up till August 12, he was bright and cheerful; but after this date he became subject to frequent attacks of causeless depression. He had had similar attacks before admission during the earlier part of his illness, but he had been free of them for three months before admission. I watched him through several of these attacks, which frequently came on quite suddenly. He would go away to an obscure corner of the corridor, or would cover his head with the sheet and cry in bed. Before admission he went out into the fields to cry. He felt as if something was going to happen to him but did not know what it could be.

He had a strong visual memory and said that when he became low spirited he saw his home "topsy-turvy, the chairs were upside down and tossed about as if there had been a row."

§ 5. On September 23 and 26 (and on other occasions when I was not present) he had a feeling of exaltation. He had been in his ordinary condition all the afternoon, and had had pain in the head, neck and shoulders. This left him at 5 p.m., and he suddenly felt very strong. He began making his bed and felt as if he could lift the whole bed from the ground. He boxed with another patient but soon saw how weak he was.

§ 6. Up to three months before his admission to hospital, he woke on several occasions to see a white figure at the foot of his bed. The face seemed covered and the figure seemed that of a person wrapped in a sheet. He could not tell whether it was a man or a woman. He was "ridiculously frightened" and sat up in bed shivering, sweating, and with "goose-flesh." He had no hallucinations of vision during his stay in hospital. His vision was $\frac{6}{6}$ in both eyes, and there was no error of refraction or abnormality in the media or fundus oculi.

§ 7. For five months before admission he had smelt curious smells that others could not smell.

On August 16, and on five other occasions during his stay in hospital, he had a definite hallucination of smell. The smell was said to resemble "bad fish," or a "drain smell" like the opening of a sewer. On several occasions it was a "fusty burnt smell." On one occasion he thought he himself stank, but neither the nurse nor the other patients to whom he applied could smell it. These smells made him feel sick and they always "seem to make the head ache very badly." On August 16 he was complaining of much temporal headache and superficial tenderness was present markedly over both temples, and to a less extent over the eyes and over the vertex. Smell was qualitatively good and the nose normal. Taste was excellent to acid, sugar, quinine and salt. The mouth was clean.

§ 8. He experienced no auditory hallucinations at any time.

§ 9. His memory began to fail four months before admission and became much worse during his stay in hospital. He used to have a particularly strong memory for names and places, and was picked out for scouting on account of this aptitude. All this power left him. He could not remember errands or orders, nor where he had put things.

He could not keep his attention on a book or a game and forgot what he read.

His memory for the remote past was unaffected.

§ 10. His temper had become very irritable within the few months preceding his admission. He would snap at the children at home and then take them on his knee, kiss them and make up to them because he felt so upset at having snapped at them. He would lose his temper with a patient and immediately go up and say how sorry he was.

§ 11. He frequently thought people were against him and when he saw two nurses talking together thought they were saying he had misbehaved himself in the ward or that he was lazy and should be got rid of. On September 25, he complained to the sister of the conduct of the nurse, but was satisfied when she told him he was wrong. He informed me that he had made this charge and said he was sure it was untrue; but "although I struggled against it, it came out in spite of me and I was very glad sister took no notice of it." He had been much troubled about these false charges he seemed compelled to make and became much happier when it was explained to him that they were a symptom of his illness.

Example 6. [Case 68, table 3.] *To illustrate the occurrence of an hallucination of vision during the course of pulmonary tuberculosis.*

James D., aged 29, single, was admitted to V.P.H., under Dr. Eustace-Smith, on February 19, and discharged March 31, 1894.

§ 1. *History.* One morning in July, 1893, he coughed and fetched up a mouthful of blood. He continued to bleed somewhat profusely for three days, and was then admitted to Guy's Hospital, where he remained for a month.

When he returned to work he was troubled by shortness of breath on the least exertion and much cough.

He had lost flesh, and occasionally sweated at night since July, 1893.

He had had no diarrhœa or vomiting.

He was a painter. He denied venereal disease, and there was nothing of importance in his past history.

His family history was free from tuberculosis, insanity, or neuroses.

§ 2. On admission he was a well-built man of 29, but looking considerably younger. He was not emaciated, but stated that he had lost weight. Anæmia and cyanosis were absent, and the fingers were not clubbed.

The temperature rose each night to between 99·4° F. and 100° F.

He was not short of breath when in bed, but became so on walking, more particularly if he attempted to go upstairs. His cough was very slight except in the morning. The expectoration was small in quantity, but contained large numbers of tubercle bacilli and some elastic fibres.

He has had pain over both supraclavicular fossæ, and in the left side over the seventh space in front and the eleventh space behind. On admission there was slight tenderness over both supraclavicular fossæ (cervical 4), and over what appeared to be the eighth dorsal in front and behind on the left side only.

He complained of headache "across the forehead," and there was some superficial tenderness in both supraorbital regions and over the vertex on the left side.

Over the right apex, both in front and behind, the percussion note was dull, the breath sounds feeble, expiration prolonged, vocal resonance increased, and râles were plentifully heard. Over the right base the note was dull, the breath sounds scarcely audible, vocal resonance good, and some superficial, irregular, crackling sounds were heard, probably due to previous pleurisy. Over the left apex the note was diminished in resonance, the breath sounds were bronchial, and vocal resonance increased, but no râles were heard. Below the angle of the left scapula some fresh râles were heard.

The circulatory system was normal, and the pulse 88.

The tongue was clean and moist, appetite good, and bowels opened daily. Neither nausea or vomiting were present.

§ 3. *Progress.* He improved somewhat, and by March 10 had lost all his pain and headache.

From March 18 onwards he lost weight, and his cough became more troublesome. By March 28 the whole right lung had become affected, and râles were audible from top to bottom. The left base was then clear. With this outburst in the right lung came on pain in the right side, accompanied by tenderness over the eighth dorsal area. The temperature oscillated between 100° F. and 101° F. and no longer reached normal in the morning.

He left the hospital with pain in the right side, vertical headache, and the right lung affected from top to bottom.

§ 4. Ever since the second week of his stay in Guy's Hospital (August, 1893), and more particularly for a few weeks preceding his admission to Victoria Park Hospital, he had occasionally thought he saw someone standing at the bottom of his bed. This figure had appeared on several occasions, and was always black. Even the face was black, and "it was not dressed in ordinary clothes, but like statues are dressed."

On the night of February 24, whilst in the hospital, he awoke to see the black figure standing at the bottom of his bed in the ward. It seemed to be "wrapped in a large cloak," was all black, and the face seemed to be covered. He was much frightened, sweated, and "came all over cold shivers." It very much upset him, and he lay awake till daylight with a bad headache over the eyes.

When I saw him next morning there was marked tenderness over the forehead in both supraorbital regions and over the left vertex.

Vision was $\frac{6}{6}$ R. and L. Jäger 1 at 5 and 14 inches. Both fundi were normal, but there was O. 5_D of hypermetropia.

This was the only hallucination of vision during his stay in hospital.

§ 5. Since he had been ill he had frequently thought he heard people walking about the house at night, and occasionally knocking at the door. This had occurred more often than once a week, and the last time was in the night before admission. These sounds are extremely unpleasant, and seem to "unnerve" him.

Watch heard at 3 feet right ear, 9 inches left ear. Fork on forehead heard best in the left ear. On examination the left meatus was found to be narrow and partly occluded by wax. On removal of this wax the membrane was seen to be healthy and not cupped. The right ear was normal.

He had no auditory hallucinations during his stay in hospital.

§ 6. At no time did he experience an hallucination of smell either before or during his stay in hospital.

Smell was good in the left, but poor in the right nostril. The septum was deviated towards the right, and he breathed with difficulty through the right nostril.

§ 7. Since his illness began he had suffered from occasional attacks of depression in which he wanted to be alone, and thought something was going to happen, but he did not know what it was or to whom it would happen.

From the first week after admission until March 25, when the right base was again attacked (*vide* § 3), he was cheerful. But he then began to suffer from marked attacks of depression. On March 28 he walked about from one place to another seeking some place where he could be alone. If spoken to he rose and went away to another part of the corridor. He had a feeling something must be happening at home, and was worried "because the home does not look the same as when I came away, but all dull and dark, and all the things seem to have been shifted."

§ 8. His memory had become very bad since his illness. He forgot where he had put his tools, and could not remember what he intended to do. He forgot what he saw in the street and had been told, especially what he had been told to do.

His remote memory was fair, and the main loss lay in memory of things that had happened during the last year.

§ 9. His temper had become very bad. He would strike the children, and then be extremely upset and sorry that he had done so.

§ 10. His attention had failed since his illness, more particularly during the few months preceding his admission. He could not keep his mind on his work, and although he used to be extremely fond of dominoes, he could not play a good game in hospital.

§ 11. During his stay in hospital with the affection of the right base, at the end of March (*vide* § 3) he began to suffer from a feeling that people were against him. His people "did not want him home." When he saw two patients talking together he thought they were saying he was not really so ill as he said he was—that he "ought to be got rid of."

Example 7. [Case 63, table 3.] *A case of pulmonary tuberculosis. To illustrate the mental changes that may accompany the rapid involvement of the base of one lung in the course of the disease.*

George R., aged 39. Admitted to V.P.H., October 16, discharged December 21, 1893 (Dr. Sainsbury).

§ 1. *History.*—Two years before admission had an acute attack ("influenza") with cough and expectoration. He stopped in bed two weeks and recovered.

Fifteen months before admission pain came on in the left side which was increased with each deep breath or cough. He lost weight and felt weak and ill.

Nine months before admission he had another attack of "influenza," with headache and pain in the left shoulder. In this attack he sweated at night and lost much flesh.

Six months before admission he began to spit blood and had been continuously ill with cough and wasting ever since.

Except that nine years ago he had "typhoid" fever with "affection of the lungs," there was nothing of importance in his past history.

§ 2. *State on admission.*—He was a large well-built man, showing distinct signs of wasting. He said he had lost 14 lbs. in the last six months. Lips and conjunctivæ were of a good colour and there was no cyanosis.

He was short of breath even when lying in bed, the cough was hard and barking, and he was bringing up a considerable quantity of expectoration tinged with blood.

He suffered from very little pain, and superficial tenderness was absent, excepting for a spot in the left supra-clavicular fossa and the maxima of D. 7.

Movement of the left apex was less marked than that of the right. Vocal fremitus was increased over the upper part of the left lung in front and behind. The percussion note was, as a whole, hyper-resonant, but was diminished over the left apex in front and as far as the level of the sixth spine behind.

Over the left apex in front and nearly to the angle of the scapula behind expiration was prolonged, vocal resonance increased and small râles were plentifully heard. Over the right apex behind, expiration was also prolonged, vocal resonance somewhat increased and a few small râles were heard.

The pulse was 80, and there were no abnormal signs in the circulatory system.

The tongue was moist and a little flabby. Appetite was good and the bowels were opened daily. No nausea or vomiting.

He was bright and intelligent, observant, obedient and easy to manage. He slept well and had had no hallucinations of vision, hearing or smell. His memory had become worse during the last nine months.

§ 3. *Progress.*—The cough steadily improved and he gained 8 lbs. in weight.

But on November 18, the cough returned and fresh catarrhal signs appeared at the left base. The pain in the left side now became very troublesome, and was accompanied by marked superficial tenderness over the cervical areas, and the third, fourth, fifth, sixth and seventh dorsal on the left side. There was also some tenderness over the cervical areas on the right side. He complained of headache over the forehead and temples and there was marked tenderness over the forehead and temple on the left side, and some tenderness over the forehead on the right side. His tongue remained clean but he suffered from nausea. He did not vomit and there was no diarrhœa.

§ 4. He began to suffer from attacks of unaccountable depression. Suddenly he would feel very miserable and did not want to see or speak to anyone. He would leave the day-room where the other patients were amusing themselves, and go to the deserted library at the other end of the corridor. During these fits of depression he was haunted by a picture of his home "all topsy-turvy as if someone had been pulling the place about." In this memory picture it seemed as if it was a dull dismal day. There was no light in the room, no fire in the grate. The bright table-cloth had gone from the table, and the antimacassars seemed to have lost their colour. On visiting day he asked his wife why she had made these alterations in the place and where she had put the antimacassars. He had a feeling he wanted to get home to put things right again.

The depression would pass off as quickly as it came, and he would wonder at the foolish things he had been thinking.

§ 5. On November 29, December 1, and December 3, he suddenly smelt an "earthy smell." He thought it was on his hands or about him somewhere, and he asked the other patients to smell his hands. On each occasion he refused his food, retched, and lost all appetite. On December 3, I was able to assure myself that there was no objective cause for this smell. His nose and mouth were normal, and the sense of smell was good. He was suffering much from headache, worse on the left side than the right, accompanied by superficial tenderness over the forehead, temples and vertex on both sides. Tenderness on the body involved the cervical areas from the third to the eighth dorsal on the left side. The temperature was 99·6°.

The signs in the chest had rapidly extended towards the left base. The bowels were opened daily, and there was no diarrhœa.

§ 6. From this time he rapidly improved, and by December 20, all pain and tenderness were gone both from the body and head. Again he gained weight rapidly, and his temperature remained below 99°. There was now dulness at the left base, with feeble breath sounds and diminished vocal resonance. Over this area small râles were heard. There were no other fresh signs in the chest.

With this disappearance of pain and quiescence of the disease after involvement of the left base his mental state again became that on admission. He was bright and cheerful, slept well and had no unpleasant dreams. No hallucinations had made their appearance since the first week in December. His memory began to improve during the last two weeks of his stay in hospital.

Example 8. [Case 83, table 3.] *A case of pulmonary tuberculosis. To illustrate the mental changes that may accompany the onset of gastro-intestinal complications in this disease.*

Frederick Thos. S., aged 28. Admitted to V.P.H. on October 12, and discharged November 25, 1893 (Dr. Clifford Beale).

§ 1. *History of present illness.*—In the early part of 1892 he began to suffer from slight cough and "rheumatic" pains in both shoulders; about two weeks afterwards pain suddenly developed in the left side. He was admitted to St. Bartholomew's hospital with cough and loss of weight. There he much improved.

In March, 1892, he again fell ill, and has been ill on and off ever since.

In November, 1892, he coughed up a mouthful of blood. He had lost 1 stone 7 lb. in eighteen months.

§ 2. *Condition on admission.*—He was a medium-sized man of 28, but looked older. There was no anæmia, but he was somewhat cachectic. Slight signs of wasting were present. The fingers were slightly clubbed.

His respiration was twenty-five to the minute and the accessory muscles of the neck were working. He suffered from shortness of breath when he lay in bed. Cough was troublesome at night. Expectoration consisted of muco-purulent lumps with much aerated mucus.

The movement of the right side of the chest was greater than that of the left. The percussion note was diminished in resonance over the left side, and behind this diminution extended as low as the angle of the scapula. At the left apex in front the breathing was cavernous and there was whispered pectoriloquy. Behind the breath sounds were bronchial, and there was bronchophony to the mid-scapular region. Râles were heard over the left side as low as the angle of the scapula.

At the right apex behind the percussion note was diminished. The breath sounds were weak and whispered bronchophony and râles were audible to the level of about the third spine.

There were no circulatory symptoms. The pulse was 96 and the heart normal.

The tongue was clean and appetite fairly good. The bowels were open about three times a day. The motions were loose and brownish yellow in colour. The urine was normal.

§ 3. On admission he complained of no pain or headache and showed no tenderness. But on October 23, he began to complain of pain over the left side of the abdomen which increased in extent and severity. The motions became fluid, light yellow and very offensive. At the same time a fresh outburst of râles appeared at the left base.

He became miserable and depressed, sitting all day with his head on his hands with a feeling that something was about to happen to him. He disliked to be spoken to, and when up sought the most lonely part of the corridor. Before he entered the hospital he had been subject to these attacks of depression, and on one occasion felt an impulse to throw himself under a train. But this passed off and until the attack of diarrhœa came on again he had been bright and cheerful.

§ 4. Before admission he occasionally woke at night to see a face at the bottom of the bed looking at him "as clearly as if it was looking through a window." It was unlike any face he had ever seen before.

§ 5. Just before admission he several times smelt a nasty smell of "burning rags." Both at night and in the day he frequently got out of bed to see if anything was burning. This never occurred until two weeks before admission.

On October 27, he suddenly smelt a smell of burnt rags. There was nothing in the ward that could account for this. His sense of smell was good to all tests. His nose was normal, tongue a little furred and breath not offensive. His temperature was 100·6° F.

§ 6. His memory had become bad lately, and he could not keep his attention fixed on anything.

His temper was very short, and from October 23 he become difficult to manage in the ward.

When he left the hospital on November 25 the signs in the lungs had extended on the left side down to the base and he had lost 5lb in weight. The temperature throughout rose at night to 100° F. or 101° F., falling in the morning to normal.

Example 9. [Case 155.] *A case of pulmonary tuberculosis which subsequently healed. To illustrate the depressed mood and suspicion that may accompany visceral reflected pain.*

Jessie A., aged 23, single. Admitted to V.P.H., September 22, discharged December 14, 1893 (Dr. Beale).

§ 1. *History of present illness.*—She was well until twelve months before admission when she began to feel weak and to have pain about the shoulders. In August, 1892, a cough began which became more troublesome and two or three months before admission the expectoration became blood-stained. She had lost weight and had sweated at night for about two months. Her menstruation had become irregular. She had had no previous illness and her family history contained nothing of importance.

§ 2. *Condition on admission.*—She was a tall girl of 23, pale but not markedly anæmic. Her temperature was 102°. Cough was troublesome at night but much less frequent during the day. The sputum contained tubercle bacilli in small numbers.

At the left apex the percussion note was diminished in resonance. Expiration was prolonged, vocal resonance increased and a few crepitant râles were heard. Behind, a few râles were heard at the left apex after coughing. She was bright, cheerful and very intelligent.

§ 3. *Progress.*—The temperature came down gradually and she much improved. But eighteen days after admission (October 10) it rose again and remained permanently over 100°. She developed pain in the right side on breathing deeply.

The right base became dull and over this dull area the breath sounds were diminished. Vocal resonance was absent and distinct friction was audible about its upper border; showing that in all probability she had developed a pleurisy with effusion.

By the beginning of November all pain had gone from the right side and the signs at the right base were stationary. But she had now begun to develop pain in the left side of the reflected visceral type with some left-sided headache. The temperature was irregular rising occasionally to over 100° at night and not always touching normal in the morning. She was putting on weight rapidly.

§ 4. From this time until the time she left the hospital she was depressed, and on December 4, I saw her in a very typical attack. She had been up and about making herself useful in the wards. But on the morning of December 4, she got up feeling tired with headache over both temples, and over the top of the head. She also had pain at the left side and at the pit of the stomach accompanied by marked tenderness. At 9.30 a.m., after she had helped to take the lunch round to the other patients a "dreadful feeling" suddenly came over her as she was sitting talking in the day room. It was a feeling "as if there was something in store for me; just the sort of feeling that went before a punishment when I was a child." She at once left the room and had hardly reached an unfrequented corner of the corridor before she burst into tears. She cried and sobbed and would speak to no one. Whenever she was spoken to she walked away to some other place. She says the feeling was one of extreme dread without anything to be afraid of and she placed her hand over the epigastrium saying that that was where she seemed to feel her fear. She had no idea what it was all about, but she told a fellow patient she was certain she must be going to receive bad news.

This depression lasted until 5.30 p.m., and passed off as rapidly as it came; but it recurred at night and again the next day.

Under ordinary circumstances when she thought of her home she pictured the sitting room bright, clean, orderly, with a bright paper on the wall, lighted by sunshine and "cheerful and nice to go to." But during these fits of depression she seemed to see her room dark as if in a fog. All the chairs were out of place and things were topsy-turvy. The whole room was filthy, and there was no one about. By the window, the box-seat with its bright scarlet cover appeared all muddy and dark. She pictured her room at the factory "all upside down and not tidy and in order as I always leave it." These two pictures haunted her throughout her depression.

§ 5. She had no hallucinations of sight or hearing in Hospital, but on one occasion had what appeared to be an hallucination of smell.

§ 6. Her memory became very bad. She forgot what she was told to do, where she had put things, what she went for, and whether she had had her medicine. She forgot what she saw, and the names or tunes of the songs at a recent hospital concert.

§ 7. She had made several friends in the hospital, but suddenly turned against two of them and accused them of speaking ill of her. She imagined they thought her lazy and that they wanted to get rid of her. She was, however, satisfied with a simple denial on their part that it was so.

§ 8. *Subsequent progress.*—I watched her for six months after her discharge from the hospital. The signs in the chest became quiescent, but she became anæmic, developed a hæmic murmur over the left base of the heart, and the hæmoglobin at one time fell to 60 per cent. of the normal. During this time the pain and tenderness spread widely, and she suffered from marked gastric symptoms.

In September, 1894, all signs of disease had gone from the chest except slight dulness at the right base. Her temperature was normal. She was no longer anæmic. All pain and tenderness had gone and she was bright and cheerful.

In February, 1896, she was well and free from pain and headache. All depression and suspicion had disappeared, and she had experienced no hallucinations of vision or smell since April, 1894.

Example 10. *To show that the pain of herpes zoster ophthalmicus may sometimes be associated with an hallucination of vision.*

George C., aged 37, seen by me at the London Hospital, August 27, 1897.

§ 1. He was well until August 21, when he began to suffer from intense pain over the right-hand side of his forehead. This pain steadily increased up till August 27.

On August 22 some spots began to come out on the forehead, and when I saw him on August 27 a well-marked herpes was present over the right half of the forehead. It did not involve the nose or the lower eyelid, and only extended backwards 2¼ inches from the centre of the eyebrow. He seemed, however, to have had pain as far back as a line passing vertically from ear to ear.

Since August 21 he had felt very ill, and had occasionally vomited; but he had no pain or tenderness in the chest, back, or abdomen. The heart and lungs were normal. The urine contained neither albumen nor sugar. There were no signs of nervous disease.

§ 2. Since the first onset of the pain he had slept badly and was troubled by horrible dreams.

In the week between August 21 and 27 he three times saw a dark figure come in at the door of his bedroom. He was extremely frightened, and went to ask his mother if she could see the figure which was visible as he sat up in bed. He had never seen anything like it before.

The cornea was unaffected and iritis was not present. Disc and fundus oculi were normal. Vision was normal and there was no error of refraction.

§ 3. The eruption gradually healed; pain left him about August 28, and he saw no more "visions."

CONTRIBUTION TO THE STUDY OF THE CORTICAL SENSORY AREAS.

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IT is with confidence of doing no great violence to prevailing views that we present for further study a working plan, already outlined in our recently published article on "Astereognosis,"[1] a plan falling in line with the views of Bastian, in that it accords a sensory function to both the Rolandic and the parietal regions, and recognises the gyrus fornicatus as a possible sensory seat.

It is true that the steady accumulation of evidence, experimental and clinical, establishing a sensory representation in the central convolutions, has somewhat overshadowed the claims of the parietal lobes to a share in this function; in fact, many authorities now regard the Rolandic region as the sole cortical seat of both muscular and cutaneous sensations. It is also true that little positive, and some negative, clinical evidence has been collected bearing upon the experiments of Ferrier, and of Horsley and Schafer on the mesial surface of the brain. The supporters of the Rolandic region generally allow, however, that the sensory area exceeds the motor, particularly in the direction of the parietal gyri. Nor are authorities wanting who still favour the sensory function of the gyrus fornicatus and hippocampus.

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[1] *Journal of Nervous and Mental Disease*, April, 1901.

Bastian,[1] in 1887, while placing the kinæsthetic centre in the central convolutions (a view now very widely accepted), still favoured the views of Ferrier regarding cutaneous sensations, a segregation of function, endorsed by Horsley and by Schafer, to which our theory practically subscribes. We would suggest, however, an anatomical and physiological relationship between those cortical areas somewhat at variance with Bastian's views as we understand them. This author, while not definitely designating the course of the fibres transmitting the impressions from muscle, fascia, tendon and skin, received by the kinæsthetic centre, conveyed the idea that all these impressions proceed directly to the Rolandic and marginal areas. He called attention to the fact, however, that extrinsic sensorial stimuli—for example, touch and smell—also play a part, adding that large tracts of the brain ought to be concerned with the registration of kinæsthetic impressions for future use in the guidance of all voluntary movements. The motor mechanism thus stimulated he placed below the cortex. In his later publication[2] (1893) he divides sensation into two varieties and gives them separate seats; the one, resulting from the execution of movements and serving to reproduce, guide and regulate the new execution of such movements, he places in the Rolandic and the marginal areas, while he places the registry of touch and general sensibility in the limbic lobe. The latter variety of sensation incites to movement, but is in no sense kinæsthetic. The former seat of sensation includes such tactile elements as have to do with kinæsthetic memories; for example, from the deeper structures.

In the plan we favour the central convolutions are not made an independent centre, but a centre assembling the various sensory memories from other parts of the cortex (for the upper extremity from the parietal lobe and for the lower extremity from the gyrus fornicatus), itself receiving directly, at the most, only the fibres conveying muscle-sense in its narrowest meaning. Nor does our plan reject, as does that of Bastian, the motor function of the cortex.

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[1] BRAIN, April, 1887.

[2] "Hysterical or Functional Paralysis," Appendix C. J. B. Lippencott Co., 1893.

A fundamental difference, again, between our plan and that of Verger[1] consists in according to the cortical centres in the parietal lobes and perhaps in the gyrus fornicatus, the less differentiated sensations of pain, of temperature and of contact which he places at a lower level.

Our proposition is based on the fact that *we need not limit the number of successive cortical neurones taking part in the reception and the transmission of sensory stimuli.* Analogy with tracts already accepted would point to the probability that several, possibly many, neurones must be interposed between the simple reception of a sensation and the assemblage of the associated memories representing the high grade of sensation which furnishes the stimulus for so-called voluntary motion.

We need only allude to this connection to the recurrence of sensory neurones in the spinal cord, medulla, and thalamic regions, a recurrence which we have no anatomical reason to believe suddenly ceases in the hemispheres, which represent merely a higher development of homologous structures. The widespread location of sensory cells in the cord, occupying, as they do, a large portion of the posterior gray matter, should also prepare us to consider favourably a scheme which involves the diffusion of cerebral sensory neurones over a large area.

Again, *a succession of neurones is acknowledged to exist in the cortical sphere of special sensation,* as evidenced by the generally accepted theory of speech mechanism, which includes, starting, *e.g.*, from the visual side (*a*) a cortical visual area in the occipital region; (*b*) a higher area in the angular gyrus concerned in mind vision; (*c*) an aggregation in Broca's convolution of the muscular and other memories concerned, known as the kinæsthetic speech centre; and (*d*) the so-called motor centres in the lower central region, for the laryngeal, pharyngeal, lingual and lower facial muscles taking part in articulation. Our theory supposes similarly (*a*) reception of simple sensations (pain, temperature, contact); (*b*) realisation of the meaning of these sensations; (*c*) an aggregation of the cutaneous and muscular memories concerned in the stereognostic sense[1] or "active touch" (including spacing, localising and position senses); and (*d*) the corresponding motor mechanism.

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[1] *Archives Générale de Medicine,* November, December, 1901.

This comparison may be represented by the accompanying diagrams.

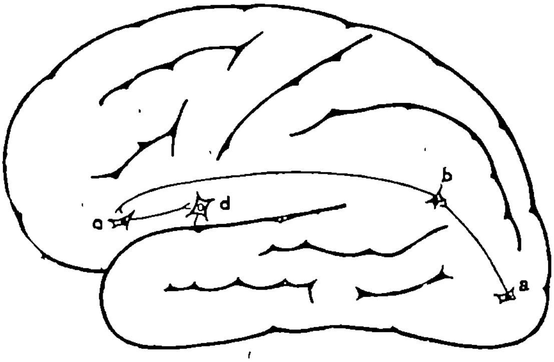

FIG. 1.

Orders of cortical neurones required for one of the paths concerned in the visual side of the speech mechanism.

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[1] This term (from *στερεός*, solid, *γιγνώσκειν*, to know), made familiar by the studies of Hoffman, Abba, Williamson, Dejerine, Sailer, Zenner, Dercum, Mills, Burr, and others, seems preferable in view of the fact that while the complete recognition of objects by the hand requires movement of the fingers, a certain degree of this sense may exist without such movement. For example, the loss of this sense can be approximately tested in the sole of the foot, without the aid even of the posture sense, by determining whether the patient can distinguish between a silver piece and a pencil applied transversely, the test being supplemented by that for the spacing and localising senses, important elements in the recognition of objects.

It has been suggested by Burr that in expressing the negative of this sense stereoagnosis is more accurate than astereognosis, but this suggestion, while well founded from a philological point of view would give rise, we fear, to much confusion on account of the similarity in appearance of the word stereognosis and stereoagnosis. The expression "stereognostic *sense*" has been also criticised and replaced by the expression "stereognostic *faculty*"; but this criticism seems to us unfounded, inasmuch as one of the legitimate definitions of the word "sense" includes recognition by the intellect. We speak, for example, of the *sense of justice*, the *sense of fitness*, and Webster quotes Tennyson's use of the term "*sense of the lover*" with a similar significance.

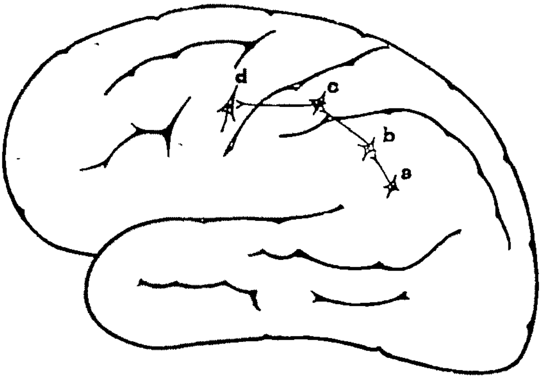

FIG. 2.

Corresponding orders of neurones, suggested as forming part of the hand movement mechanism from the point of view of one of the simple sensations contributing to the stereognostic sense.

While we do not attempt the exact localisation of the separate factors entering into this complex process, and while it seems doubtful if either clinician or physiologist will be ever in position to do so, it does seem that we are justified in placing the lower of the neurones posteriorly to the gyri in which the corresponding motor areas are found, while the highest centre must be placed in the central convolutions, closely approximating, if not overlapping, the corresponding motor region. In other words, we should favour, for the upper extremity, the middle portion of the central convolutions as the seat for "active touch," or the stereognostic sense, and the parietal lobes for the lower grade of sensory memories (contact, pain and temperature). For the lower extremity the stereognostic sense should be placed in close association with the motor leg centre. We approach the question of the lower sensory neurones for this extremity with less confidence, but it seems not improbable that the gyrus fornicatus, lying adjacent to the motor centres for this part of the body, may furnish the principal seat, though for reasons to which we shall allude later, far less satisfaction is found in the clinical study of this region than in the study of the convexity of the brain.

Regarding the location of "muscle sense" or "muscle memories," there is wide diversity of opinion. While the weight of authority favours the Rolandic region for this function, Dana,[1] for example, who favours the central gyri for all other sensory functions, places the muscular memories in the parietal lobe, a situation hard to reconcile with his idea of the unity of the cortical sensory motor memory organ. The divergence of opinion is possibly in part due to the vague interpretation of the expression "muscle sense." Fränkel wisely adopts an expression which implies merely recognition of the position of the parts without reference to the question whether this recognition involves muscle articular, or cutaneous, stimuli, or all three. We use the term "posture sense" for this reason. The muscular memories concerned in this sense we are inclined to place in the central convolutions, guided perhaps partly by our personal observations, and partly because it seems established that the fibres from the fillet pass to this region, whether directly (Fleschig and Hoesel), or with interruption at the thalamus (Mahaim, von Monakow). It seems not improbable, however, that the cutaneous and articular fibres concerned in posture sense pass first to the parietal, while the fibres from the muscle spindles pass to the central convolutions to take part in "active touch" without intervention of further neurones, though such hypotheses are perhaps too conjectural to detain us at a time when such comparatively simple functions as those of contact, pain and temperature are still under discussion.

The very frequent escape of the pain and temperature senses, compared with that of contact, in lesions of the central convolutions, renders it probable that these two senses have a separate representation in the cortex, just as their fibres run a separate course in the spinal cord. This centre may be further removed from the central region than that for touch, for in recorded cases of apparently limited Rolandic lesion the statement frequently appears that tactile as well as stereognostic sense is impaired. This does not necessarily establish the central seat of contact sense, for it is evident that many observers have used the term "tactile sense" loosely, referring only to the loss of "active touch" and even when it appears that contact sense is really impaired in these cases, the impairment is evidently slight, for the patients are described as calling two points one, as localising badly, and as being unable to distinguish between different objects, descriptions in themselves implying a certain degree, at least, of contact sense.

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[1] *Journal of Nervous and Mental Disease*, December, 1894.

We desire particularly to disclaim the suggestion that the cortical sensory tract is limited to the course we have sketched. We attempt no suggestion as to the distance apart of the cells concerned, or of the length and course of the connecting fibres, whether direct or tortuous. It is not impossible, for example, that some of the cells lie much more superficially than others, or even that the cells of some neurones directly overlie those of the succeeding neurones.

The diagram presented by Mr. Victor Horsley at the Congress of American Physicians and Surgeons, in 1888 (reproduced in Dana's article), represents three cells, one overlying the other, in the Rolandic region; lowest of all a great pyramidal cell for motion, above it a smaller cell of similar shape for muscular sense, and above this a still smaller cell of different shape for "slight tactile" sense. The supposition of Horsley that the small cortical cells are probably part of the sensory mechanism seems reasonable, and it may well be that the "active touch" area overlaps, or even extends more anteriorly than the corresponding motor centre. That *all* the sensory factors (including temperature, pain and contact) are so superimposed, however, seems to us most improbable.

We use the word "centre" in its broadest sense, meaning the physiological, not necessarily anatomical, groups of cells and fibres concerned in the process under discussion. In the case of the kinæsthetic area this group must be extensive, for it involves not only the muscle memories but also an aggregation of associative memories connected with the temperature, size, shape and weight of objects, their appearance, their sounds, the appearance and sound of their names in short it must be in connection with, and receive stimuli from every centre of sensory memories adding to our knowledge of the external world. Each of these factors plays a part in the *complete* aggregation of association memories preceding and stimulating a so-called voluntary movement however simple, for example, the closing of a window, though such movement may not necessitate the presence of all sources of the information.[1]

The recent valuable work of Cajal[2] on the brain cortex may throw light on the anatomical substratum for the kinæsthetic centres. He describes a fundamental difference in the structure of the pre-Rolandic and the post-Rolandic convolutions. The anterior of these convolutions differs, he finds, from the rest of the cortex in the following respects: (*a*) Lack of plainly differentiated granular layer; (*b*) great thickness of the layer of middle great and great pyramidal cells; and (*c*) the presence of a specific nerve plexus consisting of strong exogenous nerve fibres lying in the network of the middle great layer.

The posterior central gyrus, on the other hand, resembles, he finds, the associative cortex, in containing (*a*) a well-defined granular layer; (*b*) a very slight layer of middle great and great pyramidal cells; and (*c*) a specific nerve plexus having another seat than that occupied in the anterior central convolution. Cajal infers that either the posterior convolution has a peculiar motor function requiring a different structure from the anterior convolution, or that its motor character is questionable, and that it represents rather an ideo-motor association centre whose stimulation brings about movements in certain muscle groups. This author suggests that perhaps the small pyramidal and the polymorphous cells constitute the substratum for touch, pain and temperature senses, adding that although it is impossible to establish the true part taken by the different kinds of pyramidal cells, yet the possible hypothesis suggests itself that the pyramidal tract springs from the giant pyramidal cells and from some of the middle great pyramidal cells, while the "Balken-bahn" takes its principal, if not its sole origin in the small pyramidal cells, and perhaps also in the polymorphous cells.

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[1] The demonstration of such a kinæsthetic centre would fall in line with the inferences of Henry Smith Williams in his "Story of 19th Century Science." Harper & Brothers, 1900, p. 422.

" . . . most of the supposed isolated 'faculties' of higher intellection appear, upon closer analysis, as complex aggregations of primary sensations, and hence necessarily dependant upon numerous and scattered centres. Some 'faculties,' as memory and volition, may be said in a sense to be primordial endowments of every nerve cell, even of every body cell. . . . But such refinements of analysis, after all, cannot hide the fact that certain forms of higher intellection involve a pretty definite collocation and elaboration of special sensations. Such specialisation, indeed, seems a necessary accompaniment of mental evolution. That every such specialised function has its localised centres of coordination, of some such significance as the demonstrated centres of articulate speech, can hardly be in doubt, though this, be it understood, is an induction, not as yet a demonstration. In other words, there is every reason to believe that numerous 'centres' in this restricted sense, exist in the brain that have as yet eluded the investigator."

[2] *Studien uber die Hirnrinde des Menschen*, 2 Heft; *Die Bewegungsrinde*, von S. Ramón y Cajal. *Uebersetzt*, von Dr. J. Bresler (Leipzig, 1900).

The clinical and pathological arguments in favour of the plan suggested are:—

(1) Numerous cases have been reported with loss of the stereognostic sense, or "active touch," including loss of spacing, localising and position senses, but with preservation of pain, temperature and contact senses. This points to *a centre for the aggregation of memories concerned in judgment of the nature of objects separate from the centres whose function is limited to the simple reception of the various sensory stimuli contributing to this aggregation.*

(2) *The fact has been established beyond controversy that "active touch," or the "stereognostic" sense, has a representation in the central gyri* (Dana, Verger, and many others). Case after case has been carefully observed and reported in which Rolandic lesion has produced disturbance of spacing sense, of localising sense, of posture sense, and of the ability to recognise objects in the hand with closed eyes, as well as fumbling movements and ataxia.

(3) The temperature, pain and contact senses have been so often spared in lesions of the central gyri as to render it extremely improbable that this region is the seat of these sensations.

(4) When the parietal as well as the Rolandic region is found involved we often find, in addition to loss of active touch, loss of the temperature, pain, and tactile senses.

(5) The sensation of the lower extremity is often unimpaired in lesion of the convexity.

The additional arguments needed to establish the theory are :—

(6) Cases of limited parietal lesion in which pain, touch, and temperature senses are lost.

(Even if astereognosis should also be found in lesions thus limited, the fact would not militate against our theory, for it is obvious that disturbance of the second grade of neurone would impair the sensory pictures and thus interfere with the stereognostic sense, just as lesions of the mind vision centre interfere with speech.)

(7) Cases of limited gyrus fornicatus lesion with sensory loss in the lower extremity.

The arguments against compound sensory areas are :—

(1) Cortical anæsthesia unaccompanied by motor paralysis is of rare occurrence. (A few cases presenting such isolated anæsthesia, are, however, on record, for example, by Mills,[1] by Pierret,[2] in whose care anæsthesia persisted after motor recovery, and by ourselves.[3])

(2) The reaction time of sensation of touch is brief, midway between that of smell and that of sight (Wundt). (But the reaction time required for the *recognition of objects*, which would represent the complete sensory process involving all the organs of neurones we have suggested, would certainly be much larger than that quoted, that is, than thirteen thousandths of a second.)

With reference to arguments 6 and 7 it is most important that cases showing such limitation should be put on record when found, together with the clinical report of all forms of sensation whether such report is positive or negative. Cases of tumour limited to these areas and accompanied by careful clinical records, are far less frequently found in the literature than those affecting the central gyri. Too few cases are recorded bearing definitely upon argument 5. Deeply seated lesions, as tumour or softening in the parietal lobe, must be eliminated on account of their liability to involve either the mesial surface of the brain or the afferent fibres connecting this region with the thalamus. We should mention, however, an unusual case (hitherto unpublished), seen at the Massachusetts General Hospital in the clinic of Dr. W. L. Richardson. In this case an extensive meningeal hæmorrhage, confirmed by autopsy (Dr. Wright), compressed particularly the anterior parietal and post-central regions, the brain being found otherwise normal. This hæmorrhage caused in the arm moderate loss of contact sense and moderate astereognosis with diminution of spacing sense, but gave rise to no anæsthesia of the lower extremity. Posture, pain and temperature senses were intact in the arm; no loss of motion appeared in the arm or leg. The moderate impairment of function resulting from the pressure precludes citing the case as absolutely demonstrative of the suggestion that lesions of the convexity may spare the sensation of the lower extremity, but it is somewhat suggestive in this connection. The case is certainly worthy of record as exhibiting sensory loss, resulting from cortical lesion, without accompanying motor impairment.

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[1] *Journal of Nervous and Mental Disease*, May, 1900; *University Medical Magazine*, October, 1897.

[2] *Lyon Medical*, 31 Janvier, 1886.

[3] *Loc. cit.*

With regard to the function of the gyrus fornicatus we are greatly handicapped in our clinical and pathological study in various ways.

In the first place the *operative cases* which furnish so large a part of the Rolandic material are practically wanting, for operation would rarely be advised on a tumour diagnosticated as invading the mesial surface of the brain. It is true that many of the tumours and softenings implicating the central convolutions must of necessity impair the function of the gyrus fornicatus or its fibres either by pressure or by direct extension; from such cases, however, few direct inferences can be drawn as regards the function of the gyrus fornicatus, though the inference is patent that we should be chary of deeming such cases negative in their bearing on this region. The only other cases from which even approximately accurate conclusions can be drawn are those coming to the autopsy table. Here, again, we find dearth of material in this connection, perhaps largely on account of the lack of that special interest on the part of the clinician which leads him in case of Rolandic lesion to first make careful and complete clinical records, then follow the case to *post mortem,* and finally to put it on record in the literature. Again, the sensation of the lower extremity is less often carefully studied than that of the upper, a fact still further narrowing the group of cases furnishing light on this branch of the subject.

In illustration of this lack of material we need only contrast the twenty limbic lobe cases collected by Dana[1] in 1888, with the 142 Rolandic cases collected by the same author at the same date. Of even these twenty cases thirteen offer little aid as regards the gyrus fornicatus because the lesion only involved the gyrus hippocampus, fusiform lobe, or unsinate gyrus.

In four others of the twenty cases the lesion was described respectively as limited to the anterior half, the anterior third, the posterior part, "very limited" and "without any general anæsthesia," the last case being reported from a verbal communication, too vague, therefore, to be accorded serious consideration in a critical analysis.

In the remaining three cases involving the gyrus fornicatus it appears that numbness *was* present in some degree at least, for in one case numbness is reported *in the foot,* in another the statement is made that no *marked* anæsthesia was present, while in the third case the numbness was attributed to the fact that the lesion included also the central region.

The innumerable cases of Rolandic lesion on record as free from disturbance of sensation receive scant consideration at the hands of this author, and justly so, on account of (*a*) incomplete observation; (*b*) late observation after compensation has occurred; (*c*) small lesion; and (*d*) slowly developing lesion. If the same reasoning is applied to these twenty cases involving the mesial surface of the hemisphere, we are left with little or no pathological evidence worthy of serious acceptance, which negatives the gyrus fornicatus as a sensory area. It is also true, on the other hand, that we have as yet little satisfactory clinical evidence corroborating the experimental evidence in favour of this region.

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[1] *Journal of Nervous and Mental Disease,* October, 1888.

Before reviewing the more extensive, and, it seems to us, satisfactory evidence in support of the first four arguments advanced, we may be pardoned for a digression, bearing upon the difficulty of collecting the clinical material for these studies.

Research based upon recorded cases is baffling at the best, on account of (*a*) *the vague localisation of lesion* as recorded by the pathologist (for example, "under the parietal boss"); (*b*) on account of *the uncertainty whether the anæsthesia results from the lesion described*, or from pressure, œdema, or from other influence referable to remoter regions; and (*c*) *incomplete and inaccurate clinical records*.

The second and third are the most frequent and troublesome of these drawbacks. Comparatively few of the many recorded conform even approximately to the requirements of Hale-White[1]—limited lesion, definitely localised, careful autopsical record, careful clinical study. The post-operative cases are peculiarly misleading. In illustration of the *extent of post-operative symptoms* as compared to those resulting from the original lesion may be cited the case of Albertoni and Brigatti (*Revista Sperimentale de Fren.*, xix., January 1, 1893, abstract by Dana in *Journal of Nervous and Mental Disease*, December, 1894). In this case the tumour, the size of a hen's egg, was removed from the right central convolutions. Tactile, general, thermal, painful and muscular sense impressions, previously intact, were, after operation, affected over the whole left half of the body except the face.

In the case reported by Williams (*New York Medical Journal*, January 9, 1882, also abstracted by Dana), a compound fracture of the skull in the arm centre accompanied by laceration of the surface of the brain and slight loss of substance is said to have produced before operation, no disorder of sensation, though the arm below the elbow was completely paralysed. After recovery from operation, paræsthesia appeared in the left arm and leg and later examination showed persistence of anæsthesia of the fingers. Even here the character of the anæsthesia tends to support our contention, for the thermal and pain senses were intact, though articles were picked up with difficulty and in a fumbling manner, and the patient could not distinguish between a knife and a pencil held in the hand with the eyes closed.

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[1] *British Medical Journal*, July 29, 1893.

Again, in the second of our personal cases, the removal of a bit of diseased cortex in the hand area was followed by an aphasia which limited the vocabulary to the words "yes" and "middling," with marked word deafness and mind blindness, the patient understanding only the simplest orders and being unable to imitate even the simplest motions. The sensory symptoms which accompanied the motor paralysis in this case prior to operation consisted in loss of recognition of objects in the hand with the eyes closed, impairment of posture sense, localising sense, spacing sense, and pressure sense, but no impairment of contact, pain, or temperature senses. The only indication of aphasia before operation was a moderate difficulty in finding words, no mind blindness nor word deafness. The post-operative sensory aphasia precluded detailed examination of sensation after the operation, but it is obvious that whatever results might have been obtained by such examination could hardly have been attributed to the original limited lesion in the central convolutions.

Finally, in a case mentioned by Verger (reported to him by Rocher), no sensory loss was caused by an abscess in the Rolandic region causing hemiparesis, but after its evacuation, active touch, position sense, and localisation of pain were lost. Even here the preservation of temperature, contact and pain senses brings this case in line with those we shall adduce to substantiate the theory of separate representation of the latter senses.

With regard to the *incomplete and inaccurate records*, a large proportion of reported cases must be discarded in the critical analysis on account of the meagre description of the sensory loss. Description limited to the words "anæsthesia of arm," "numbness of arm and leg," while useful, perhaps, in showing that cortical disease leads to sensory as well as motor impairment, are of little aid in determining the exact location of the different qualities of sensation. Even among the more carefully recorded cases there appears a tendency to use the word "tactile" in the vague manner to which we have alluded. For instance, Dana states of a certain case that the tactile sense is impaired in a case of Rolandic lesion with marked astereognosis, though in recording the defective spacing sense he states that two points widely separated *were felt as one*. It is obvious, therefore, that in this case "contact" sense was preserved, to a certain degree at least, and we are left in doubt whether it was definitely tested, or whether the loss of so-called tactile sense was limited to the defective judgment regarding the contact, in other words, to the sense of "active touch."

Before leaving this branch of the subject we must also remember that *these investigations are pursued upon a class of patients often unable* (whether naturally or from the cerebral disease) *to understand the questions put to them*, and to analyse their own sensations. In illustration of this difficulty we need only cite a case recently examined, but not published, by ourselves. This was a case of an uneducated woman about 60 years of age. She could neither read nor write, but possessed the average intelligence of her class. Right hemiplegia existed of six years' standing, with practical recovery of the right leg, but marked athetoid movements of the right arm. There was no paralysis of the face, and the patient found no difficulty in ordinary conversation. Posture sense even as high as the wrist was absolutely lost. Objects placed in the right hand (a purse, a knife, a pencil) were unrecognised with the eyes closed, whereas in the left hand they were promptly named. The temperature sense was unimpaired in the right, as in the left hand, the patient promptly distinguishing between the test-tubes of cold and of hot water and named them correctly. On analysis of the pain and tactile senses her answers were most misleading, apparently through lack of appreciation of the meaning of our questions. For example, when touched with a pointed object in the palm of the hand (with the eyes closed) and asked if she felt it, she invariably answered "no," and yet when she was touched with two points and asked to state whether she was touched with one or two, she always answered promptly "one point," adding, "it hurts," "it's sharp," meantime drawing her hand away quickly. When touched with the same sharp point in the same manner, and asked if it hurt, she invariably answered "no." It seemed apparent therefore, that both pain and contact senses were present, notwithstanding her answers. This case is mentioned in this connection merely to show the difficulty of examining persons with defective intelligence, but not as absolutely demonstrating the persistence of contact and pain senses. For the latter purposes this case would be of no material assistance as we have no means of demonstrating the limits of the lesion.

Such considerations as above outlined must be borne in mind in every attempt to analyse recorded cases of defective sensation in cerebral lesion, and must lead to the elimination of very many of the tabulated cases.

It is hardly necessary to quote individual cases from the large number which corroborate the first of the arguments advanced in defence of our position, that is, the constancy of astereognosis in Rolandic lesions. Among the twenty-five personal and other cases contained in Dana's most recent collection (1894), wherever mention appears of active touch or its components, in connection with limited Rolandic lesion, this sense is found defective. The same is true of the collected cases of Verger. Four such cases coming under our own observation were published in our previous paper. In opposition to this view Mills[1] states that innumerable cases of Rolandic lesion have been recorded without loss of sensation. Our own study of such records would indicate that the stereognostic sense had not been as a rule investigated. Such cases do tend, however, to substantiate the claim that the touch, temperature and pain senses have a different seat, for some or all of these are the senses ordinarily tested, and in fact they are sometimes mentioned in the negative cases alluded to, for example, in one of the cases reported by Hoppe.[1]

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[1] "The Nervous System and its Diseases," Lippencott, 1898, p. 341.

With regard to the frequency with which temperature and pain senses remain intact in lesions limited to the Rolandic area, we find, taking from the same collections those whose definiteness of location and careful clinical records justified their use (Knapp, Williams, Dana, Steele, Thomas, Starr, Mason, Bremer and Carson, Darkschewitsch, Albertoni and Brigatti, Frank and Church, Laycock, Verger, Verger and Bougard, Rocher, Raymond, Wernické) that the temperature sense was normal in about half the cases in which this sense was recorded, and the pain sense in more than half of the corresponding cases. The "touch" sense was normal in several instances, although astereognosis was present. The number of cases analysed was too small for statistical purposes, to say nothing of the fact that in these selected cases we have included the Rolandic case of Darkschewitsch, assuming that he included pain, touch and temperature in the statement that all forms of sensation were lost. Again, either view might be taken of the case of Albertoni and Brigatti, in which a large tumour in the Rolandic region is recorded as producing no loss of these senses before operation, but of their affection after it, obviously the result of the operation rather than of the tumour.

The results of this analysis is given therefore, merely as suggestive and for comparison with the cases known to be extensive, but without claim of statistical accuracy. This variety of research is discouraging on account of the laxity in sensory records and the indefinite knowledge of the lesions. The latter must of necessity always offer a serious obstacle, but it is to be hoped that the former will disappear as interest in this subject becomes more general.

Our personal cases, in which the physical examinations were made with special reference to the questions under discussion (published in detail in our previous article), would certainly indicate that preservation of touch, pain and temperature senses with absence of the stereognostic sense is common in Rolandic lesion, and before the view of Mills is accepted (namely, that the stereognostic sense has its seat in the parietal region) the burden of proof should be sustained by at least matching such cases by cases in which the stereognosis was the only sensory symptom resulting from parietal lesion.

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[1] *Journal American Medical Association*, February 2, 1901.

Case 1.—Young woman. Numbness of left arm, six weeks' duration. Examination of sensation of upper extremity showed astereognosis with loss of position, spacing and pressure, slight impairment of touch, and *preservation of temperature and pain senses.* Five days later hemiplegia appeared. Operation on arm area in the Rolandic region. Evacuation of cyst. Death after one month. Autopsy refused.

Case 2.—Young woman. Numbness of right hand one month ago, followed in a few days by loss of power. Several days later loss of power and sensation in right leg. Upper extremity showed impairment of stereognostic sense and lessening of pressure, position, spacing and localising senses. *Preservation of contact, temperature and pain senses.* Headache, vomiting, optic neuritis. Knee-jerks increased on both sides. Babinski reflex on the right.

Operation.—Discoloured area size of half-dollar in Rolandic region. Removal of small fragment only. Operation followed by marked aphasia, large cerebral hernia, relief of pressure symptoms, but no amelioration of sensory or motor symptoms.

Case 3.—Young woman. Convulsions two years ago followed by paralysis of left leg. Gradual increase of paralysis with spastic symptoms. Repeated Jacksonian convulsions, spasm always beginning in great toe and followed by temporary weakness in left arm. Face unaffected. Optic neuritis. Both knee-jerks increased. Ankle clonus and Babinski reflex on left. No loss of sensation in hand. In foot loss of spacing and posture senses. Patient could not distinguish between silver piece and pencil applied transversely to sole of foot. *Pain, temperature and contact senses normal.*

Operation.—Just behind upper end of fissure of Rolando. Brain tissue discoloured, bulging, vascular. Hæmorrhagic focus from which old clot, size of thumb, removed. Diagnosis from bit of cortex removed, red softening. After operation same loss of sensation with added slight blunting of pain and contact, not of temperature sense.

Case 4.—Woman, aged 92. Twelve days before examination developed choreiform movements in the left arm, gradually spreading to face and leg of same side. Strength not notably impaired. Grasp good on both sides. All voluntary movements made, but in an incoordinate manner. Loss of stereognostic sense in left hand and foot with loss of spacing, position and localisation senses, but there was *preservation of contact, pain and temperature senses.* Death six days later. No autopsy. (This case illustrates the dissociation of pain, temperature and contact senses from those of spacing, localising and "active touch," and while the seat of lesion was not verified by autopsy, there can be little doubt that it was superficial and in or near the Rolandic region.)

In all four of these cases astereognosis was present, therefore, with preservation (before operation) of temperature and pain senses, and in three of the four contact sense (tested with special care), was found normal, and in the fourth was only moderately impaired. The fact that in one of the cases after operation slight blunting of pain and contact senses appeared does not impair our conclusions in view of the common post-operative extension of symptoms.

This result certainly suggests that the representation of the temperature and pain senses and probably of the tactile (aside from active touch) lies mainly outside of the Rolandic area. Indeed, when we bear in mind the prevailing laxity in the use of the word "tactile," and the possibility, even probability, that in some, at least, of these cases the limited lesion mentioned was not responsible for all the symptoms, we feel that these cases give rise to a strong presumption in favour of a separate seat for the sensations at least of pain and temperature, and probably also for that of contact. This presumption is fortified by the study of cases in which the lesion described was more extensive, including, for example, the parietal lobes, the angular gyrus and the occipital region, and extensive implication of the centrum ovale.

Of such cases we find in the same collection four. In all these cases there was loss of touch and pain sense, and in three of the four, loss of temperature sense. In the fifth this sense was not mentioned. As regards these senses, the cases of extensive lesion offer, then, marked contrast to the cases of limited Rolandic lesion. The cases are those of Madden[1] (post-central, superior parietal, suprasylvian, anterior portion of angular gyrus and occipital convolutions. Loss of *pain* and *touch,* temperature not mentioned); of Wilson,[2] (tumour of central region with softening of tissue around the tumour and white softening of whole centrum ovale, *loss of pain, touch* and *temperature*), of Dejerine and of Long. The following were the essential features of the two last mentioned cases:—

Case of Dejerine (*Rev. neurologique,* Mars 15, 1893, p. 51).

The lesion consisted of softening in the inferior parietal, the central, the first and second frontal, and first temporal gyri. There was (left) hemiplegia of the face, arm and leg, the arm being more affected than the leg. In the arm there was complete *loss of contact, pain* and *temperature* senses. In the lower extremity the same senses were less plainly felt than on the other side, but were "far from being abolished." Muscle sense lost in arm, very slightly in the leg.

Case of Long (*These de Long,* observation 54, p. 268).

The lesion was a plaque occupying the foot of the second frontal, the lower part of the Rolandic convolutions, all of the inferior parietal, and the angular gyrus; the anterior part of the occipital and the upper and posterior part of the second temporal and part of the first temporal lobes were also involved. There was *notable diminution of the tactile, pain* and *temperature* senses in the hand; the muscle sense was extremely altered.

The case of Hudson[3] apparently falls between the limited and the extensive classes, in that the traumatic meningeal hæmorrhage ¾ inch thick, removed from the central region, extended, according to the diagram, beyond the limits of the central gyri. In this case astereognosis was marked, temperature and pain senses were impaired but not lost. Touch sense aside from active touch was not noted.

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[1] *Journal of Nervous and Mental Disease,* February, 1893, p. 125.
[2] *Lancet,* February 27, 1892, p. 482.
[3] "Annals of Surgery," 1893, p. 421.

The following (hitherto unpublished) personal observation though unverified by operation or autopsy, is of great interest in this connection.

Case 5.—Man, aged 72, with marked atheroma. Intermittent twitching of the right side of the face first appeared five weeks before examination. Each attack lasted a few minutes, and the frequency had increased to about seven a day. Three weeks before examination a similar spasm of the right hand appeared. These attacks were followed by motor paresis of face and hand. No headache, no nausea nor vomiting, no optic neuritis.

Sensory examination showed complete loss in the hand of all senses, including contact, pain and temperature, the anæsthesia being bounded sharply on the palmar surface by a transverse line just above the wrist, on the dorsal surface by a similar line just above the knuckles.

In the lower extremity there was *no loss of temperature, contact, or pain sense*, but there was marked loss of spacing sense in the sole of the foot, and the patient was unable to tell the difference between the point and side of a pencil. Tactile hyperæsthesia accompanied the astereognosis. The gait was normal and all movements of the foot were made, the knee-jerk was active but not markedly exaggerated; there was no Babinski reflex. The grasp was normal, but the movements of the right hand were awkward. The patient was able to make the necessary movements for touching the thumb and little finger, but was unable to guide them even with the eyes open. Typical right facial paralysis was present with drooling, difficulty in exhibiting teeth, thickness of speech and deviation of the tongue to the right. It was hard to choose and use words, but there was no sensory aphasia.

The difference both in type and degree of the anæsthesia in the upper and lower extremity was striking in this case. The limits of the lesion, probably softening, are, of course, uncertain. but the suggestion forcibly presents itself that the escape of numbness, other than astereognosis in the foot, was due to the integrity of the mesial surface of the brain, and that the complete loss of pain, temperature, and contact senses in the hand, pointed to involvement of the parietal, as well as the Rolandic, convolutions.

In conclusion, though we have not yet established our theory, it seems to us that we have offered presumptive evidence of sufficient weight to justify taking it into consideration in the further study of this subject. Especially desirable are cases of limited parietal or gyrus fornicatus lesion accompanied by careful clinical records.

We would particularly emphasise the importance of recording the sensations of both extremities in every case of supposed cerebral lesion. In making such records it is important to include (1) the temperature sense; (2) the pain sense; (3) contact sense, that is, the ability of the patient to recognise the *fact* that he is touched; (4) the localising sense, including localisation of pain; (5) posture sense, including both, noting (*a*) whether the patient knows that the part is moved, and (*b*) whether he knows the direction in which it is moved; (6) spacing sense; (7) pressure sense; (8) stereognostic sense, or ability to recognise objects in the hand with eyes shut. (This sense may be approximately tested in the sole of the foot by applying alternately the end and the side of a pencil. If they are distinguished, and if the localising and spacing senses are normal, there is no marked loss of the elements furnishing the stereognostic sense in this extremity.)

That such systematic examination will be rewarded is evident from the fact that since the introduction of the study of astereognosis the symptom has been found by Verger in more than half the recent cases of hemiplegia; by Abba in 8 per cent. of fifty old and recent cases; by Darcum in nearly half of forty-one cases; and by ourselves in 33 per cent. of seventy-three old cases.

It is also desirable that more pains should be taken to investigate in such cases the sensation of the face. No study has been made, as far as we are aware, of "active touch" in the face, and yet the tongue, at least, possesses this function in a high degree. In a case of astereognosis of the right hand, recently seen in consultation with Dr. Phippen of Salem, cloth could not be distinguished from paper on the right side of the tongue though easily distinguished on the left. Contact sense was unaffected.

This case showed also the importance of testing the ability to handle tools and to write, for the patient, though able to make every movement, and though able to talk fluently, had lost the power of sewing and of writing (except by slowly tracing the letters, by which method she wrote spontaneously and from dictation). This observation tends to show that the stereognostic hand-centre is closely identified with the general kinæsthetic hand-centre, and further, that our search for the writing-centre need not lead us beyond the network subserving these functions. There certainly seems every reason to accept Bastian's suggestion that the cortical structures in which "are registered the sensory impressions produced by writing movements," though functionally related, need not have a separate seat, but may be mingled with those structures concerned in the memories of other hand movements. The existence of a writing-centre in this sense can hardly be doubted, whether its perfect action requires the integrity of Broca's region, or whether it is capable of acting independently of that region in response to appropriate stimuli from the auditory and visual centres.

A CASE OF
CHRONIC INTERNAL PACHYMENINGITIS OF THE SPINAL CORD.

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THE following case seems worthy of record as an example of a rare form of disease. In this particular instance a most striking feature is the extent of the disease. In most cases of chronic meningitis the lesion affects only a limited extent of the cord, being most frequent in the cervical region. Here the meninges are affected the whole length of the cord, with the exception of the extreme ends. The disease appears to have started in the lower dorsal or dorsi-lumbar region, and thence to have gradually extended, the arms being seriously involved for only a short time before death, long after paraplegia had been established. But although the spinal meninges were so extensively affected, the disease remained absolutely limited to the spinal canal, and did not at all involve the intracranial membranes. The case also shows the important part in the symptomatology of the disease to be attributed to the destruction through pressure of the nerve-roots. The cord itself showed extensive changes, probably secondary, and due chiefly to compression, chronic venous congestion, and increase of its interstitial framework. The symptoms in order were pains, gradual loss of power, at first in the legs, then in the arms, bladder-troubles, affection of sensation, muscular wasting, and contraction, with formation of bed-sores.

The patient, Charles C., unmarried, aged 25, was a Dorsetshire shepherd. He came of a healthy and long-lived family, and his parents were living and well. He had rheumatic fever at the age of 6, after this he had apparently rheumatic iritis, and lost the sight of one eye; he had a second attack of rheumatic fever in the same year, and a third at the age of 21. He was able to work as a shepherd, and his occupation involved much exposure; he frequently got wet through and remained in his wet clothes all day. He positively denied syphilis and gonorrhœa, and had always been temperate. He attributed his illness to rheumatism and exposure to wet.

The present illness began rather suddenly about eighteen months previous to admission to the Bristol General Hospital, with loss of power in the legs, and severe pains which were attributed to rheumatism. Since then he has done no work, and has been confined to the house or to his bed. He has hardly ever been free from pains of a dull aching character in the ankles and knee-joints, and in the wrists, elbows and shoulders. The joints frequently swell and become hot and red. The pains are fugitive in character, sometimes dart through the limbs, and also affect the muscles in the neighbourhood of the joints, which are the seat of spasm. The pains are worse at night and after any exertion. He has gradually lost power in his legs, and occasionally has incontinence of urine. He suffers from shortness of breath, and swelling of the ankles.

On admission, January 27, 1900, the man looked considerably older than his years; he was fairly well nourished, but the muscles were everywhere flabby. There was a marked fine tremor of the limbs, and some fibrillary twitchings of the muscles. He could not stand without support, and when aided could just walk a few steps with a shuffling gait, dragging his feet. He spoke slowly and deliberately, was slow in answering questions, but appeared cheerful, with a sense of well-being remarkably in contrast with his physical condition. The pulse was small and weak, the tongue tremulous, the temperature normal, the bowels constipated.

The heart was enlarged, the præcordial area bulging, the apex beat was in the sixth space, one and a half inches outside nipple; over the mitral area and conducted into the axilla was a loud, rough systolic murmur.

Otherwise the thoracic and abdominal viscera were normal, and the urine contained no albumen.

The cranial nerves were unaffected.

The arms were weak but not paralysed; they were agitated by a constant fine tremor even when lying at rest. He had pains in the arms but no loss of sensation.

The legs were very weak, the muscles somewhat wasted, the toes were in position of extension, and dorsiflexion of the foot was very feeble; he could raise the legs off the bed, but had difficulty in flexing the knees and hips, the flexors being weaker than the extensors. There was slight loss of tactile sensation and of temperature sense over the legs generally but it was not very definite at this date. He complained much of severe pains in the knees and of tingling in both legs.

There was some lordosis and stiffness in movement of the lower dorsal and lumbar portions of the spine, and slight tenderness on percussion of the lumbar spines. He complained of a girdle pain at the level of the umbilicus.

There was some hyperæsthesia of the skin of the left side of the trunk.

Reflexes.—Abdominal and cremasteric absent, plantar present; tickling of the left sole caused contraction of the left quadriceps extensor.

The knee-jerks were exaggerated, ankle clonus was not present.

Elbow and wrist-jerks were present.

He had retention of urine with overflow-incontinence.

There was a large bed-sore over the sacrum, and another over the great trochanter of the right thigh.

The symptoms, on admission, were thus rather indefinite with regard to diagnosis. He at first improved a little in some respects, probably because he had not received proper attention before admission, and he lost the marked tremor of the muscles. There was, however, no real improvement, he suffered from a severe girdle-pain, and from intense pains in the limbs, worse in the legs than in the arms. The leg-muscles rapidly wasted, and the bedsores, although the sloughs separated, showed no tendency to heal.

On February 20, the leg-muscles showed much wasting, there was some rigidity of the left lower extremity, the left knee-jerk was much exaggerated. Other reflexes as above. The left leg was completely paralysed, but there was still very slight power of movement in the right.

The condition of sensation, so far as it could be accurately determined, for the patient gave contradictory answers in this respect, was briefly as follows. A zone of hyperæsthesia, three to four inches wide around the trunk at the level of the umbilicus. Hyperæsthesia to pin-prick over the front of the thighs, over the front of the legs and, in places, over the calves. Anæsthesia to pain over the dorsum of the feet, and on the outer aspect of the legs; patches of anæsthesia, the outlines of which could not be accurately determined, over the trunk below the umbilicus, on the outer side and back of the thighs, and over the buttocks, and in places over the calves. The temperature sense was very defective or lost in places over the lower extremities, and there was also some deficiency of tactile sensation, varying in different parts, and also at different examinations. The chest, upper part of back, shoulders, arms and neck showed no affection of sensation, but over the elbows there was some hyperæsthesia, and tactile sensation was blunted over the left hand and forearm.

On February 25, there was loss of power in both lower extremities which were rigidly contracted, and flexed at all joints; the wasting of the muscles was more marked, the calf muscles being especially affected; the most wasted muscles gave no reaction to the faradic current. Loss of sensation to the prick of a pin seemed almost complete over the legs. The pains in the joints were very severe, and the girdle pain very troublesome. Owing to the contraction of the legs the knee-jerks could no longer be obtained. During the last few days he had complained of tingling and numbness in the right arm, though no objective loss of sensation could be made out, and he now entirely lost power in this limb, which lay helpless by his side, the muscles were flaccid, and large bullæ appeared over the outer side of the right thumb and on the index-finger.

On February 28 the left arm began to be affected. The patient, during the last week, had become absolutely helpless and powerless to move himself; he lay on his right side with the legs rigid, wasted, and drawn up, the right arm flaccid, and with only power to move the left arm very slightly, and to feebly turn the head and neck. There was incontinence of urine and fæces. The abdomen became very much distended and tympanitic from paralysis of the abdominal muscles; the movements of the diaphragm were very feeble, those of the intercostals fairly good.

One of the most marked clinical features of the case during the last three weeks of his life was the rapid formation of bedsores, which formed over every bony point on which there was the least pressure. Thus they occurred over the sacrum, trochanters, bony points of the knees, heels, great toes, outer surface of feet, thumbs, elbows, wrists and shoulders, rapidly forming on every bony angle or prominence, in spite of everything that was done to prevent them. As a consequence he suffered from septicæmia, and died on March 25.

The bed-sore over the right hip exposed the bone, and was the most extensive that I have ever seen. The pains in the limbs continued up to the last, a[illegible]re worst in the legs; he had a feeling of a band round the right arm, and the girdle pain shifted during the last week from the abdomen to the upper part of the chest. For the last three weeks of his life the only voluntary movements left were those of the face and eyes, of swallowing and speech, and ability to slightly bend the head to one side or forwards.

At the *post-mortem* examination bed-sores were found in the positions above described.

The lungs were œdematous, and the lower lobes the seat of hypostatic pneumonia. The bronchi were filled with frothy mucus. The heart showed old disease of the mitral valve; its segments being much thickened, opaque, and somewhat contracted; the left ventricle was enlarged. The liver was pale, and showed slight fatty infiltration. Spleen slightly enlarged. The kidneys were deeply injected, surface mottled; capsules stripped well; cortex pale between the injected vasa recta. There was chronic catarrh of the bladder, and the ureters were a little distended.

The skull was normal. The cranial dura mater was healthy, the pia arachnoid a little opaque, but not distinctly thickened. The cerebro-spinal fluid was in excess, and the cerebral hemispheres small, but appeared healthy on section, except that the ventricles were much dilated, especially the lateral ventricles, and the ependyma somewhat thickened. The cerebellum, pons, medulla and their coverings were healthy.

The brain weighed 40 oz. The cerebral convolutions were normal in arrangement. The vessels at the base appeared healthy. No other morbid changes were found in the viscera, and it may be especially mentioned that no gummata or other evidences of syphilis were found. The bones of the spinal column were normal, and externally the dura mater appeared healthy throughout, except for some injection of the vessels on its surface; it was nowhere adherent to the spinal column.

On removing the cord and its membranes, and making transverse sections, a remarkable condition was manifest. The cord for its whole length was enclosed in a tough fibrous case or sheath. This sheath consisted of the greatly thickened dura mater, and within this of a thick layer of looser connective tissue. The whole was firmly adherent to the spinal cord on its dorsal and lateral, less firmly on the anterior aspect. The thickening of the membranes extended the whole length of the cord, being greatest in the lower cervical and in the dorsal and lumbar regions. It gradually lessened in thickness at the upper part of the cervical region, and below at the lower lumbar and the sacral regions. The thickening ceased below the foramen magnum and did not extend on to the medulla, nor did it involve the cauda equina. It extended right round the cord, completely enclosing it in a fibrous casing, but the thickening was greatest over the dorsal aspect, where in the seat of its greatest development it attained a thickness of a quarter of an inch. The cord itself appeared small and compressed.

The cord and its membranes was hardened in Müller's fluid, and sections were cut from all parts of it after embedding in celloidin. The sections were stained by the Schäfer-Pal method in hæmatoxylin-eosin, by van Gieson's method in methylene blue and aniline blue-black. Small portions of the cord and membranes were also hardened in alcohol. Sections from these were stained in Löffler's methylene blue and in gentian violet for micro-organisms. No organisms were detected in any of the sections.

Microscopically the exudation was found to consist most externally of the greatly thickened dura mater, and this part was composed of closely applied layers of dense fibrous tissue. Between the layers were large venous spaces filled with blood, but this part was not so vascular as the next. Beneath this layer and next to the cord which it surrounded was a thick layer, constituting about two-thirds of the whole exudation, of a looser, less dense texture than the preceding, composed of finer, more delicate connective tissue in lamellæ and interlacing bundles; this layer was very vascular, showing a large number of deeply injected vessels, and enclosed the nerve-roots which traversed it. Within this and immediately surrounding the cord was the pia mater, which was thickened and its vessels injected.

As said above, the mass of connective tissue was thickest on the dorsal and lateral aspects of the cord.

Nerve-roots.—Both anterior and posterior roots were compressed and largely destroyed by the exudation, their connective tissue framework was greatly thickened, and the vessels running into the roots were distended with blood, and very conspicuous under the microscope. The Schäfer-Pal sections showed that both anterior and posterior roots contained a large number of degenerated nerve-fibres; in those most affected about one-half or two-thirds of the nerve-fibres had disappeared.

The posterior root ganglia also showed marked changes, consisting in atrophy and destruction of the ganglion cells, and a large overgrowth of the interstitial connective tissue, with injection of the vessels.

The degree of affection of the nerve-roots and ganglia varied somewhat in the different parts of the cord, according to the development of the exudation, but they were extensively affected in sections taken from all levels of the cord. A curious feature in the Schäfer-Pal sections in the most affected parts of the cord was the presence under the thickened dura mater of lymphatic spaces filled with fatty material stained black by the osmic acid, apparently due to fat carried off from the destroyed medullary sheaths.

It should be added that both anterior and posterior roots were especially compressed in the denser tissue of the thickened pia mater just at their exit from and entrance into the cord.

Spinal cord.—As to the cord itself it was closely surrounded, as said above, by the thickened pia mater, and the processes which enter the cord from this were much thickened. The short vessels entering the periphery of the cord were deeply injected.

Sections stained by Schäfer-Pal method showed a circumferential rim about one-eighth of an inch in diameter which was devoid of nerve-fibres; the zone of white matter just internal to this rim contained fewer medullated fibres and more interstitial tissue than the underlying parts. There was a diffuse degeneration of quite irregular distribution, most marked in the posterior columns and next in the middle of the lateral columns, due to increase of interstitial neuroglial tissue and atrophy of nerve-fibres.

There was no degeneration of any recognised tract of fibres in the white matter in spite of the destruction of the posterior roots. In the gray matter, in sections stained in this way, there was a great diminution in the number of the fine and large medullated fibres normally present; and the commissures showed in a marked way the decrease in the number of healthy nerve-fibres. In sections stained by other methods the circumferential rim of degeneration was seen to be occupied by increase of the neuroglial tissue and overgrowth of the processes passing in from the pia mater. The general increase in the interstitial tissue both in the gray and white matter was very marked, and the nerve-cells in the gray matter in all parts were in large numbers in different stages of atrophy and destruction; in many sections the large cells of the anterior horns were hardly distinguishable. The vessels in all parts of the cord were distended and full of blood, varying however in this respect at different levels; these vessels within the cord were not noticeably affected by the endarteritis found in the vessels in the thickened membranes.

In the latter the arteries showed very marked thickening of their coats, the arteries lying in the pia and in the loose tissue intervening between that and the dura mater being especially diseased. The adventitial sheath was moderately thickened, the muscular coat unaffected, the chief change being in the inner coat, which showed very marked endarteritis. The increase in this coat was due to great overgrowth of the fine connective tissue internal to the elastic layer, and there was also some proliferation of the endothelium. This increase in the intima occurred quite regularly around the whole circumference of the vessel, and showed no bulging into the lumen at any one point, but produced a concentric narrowing of it.

With regard to the pathology of the case similar changes in the membranes are known to occur as the result of syphilis, but these usually affect a limited portion of the cord, most commonly its cervical region. The remarkable feature of this case lies in the implication of the whole length of the cord. The changes in the cord itself seem to be entirely secondary, and due to direct compression by the thickened membranes, to intense congestion of the vessels, to extension into the cord of connective tissue from the processes of the pia, with overgrowth of the interstitial framework of the cord and consequent atrophy of the nerve-cells and nerve-fibres, and especially to extensive destruction of the anterior and posterior roots. There was no evidence of a primary myelitis, or of patches of myelitis occurring coincidently with the meningitis, and there was nowhere anything like a gumma, nor any caseation of the newly-formed tissue. The whole process was evidently a very chronic one. The changes in the vessels would be in favour of a syphilitic origin of the disease, and this is the view that I am inclined to take of it. One must admit, however, as against this view the negative history, and the absence of other syphilitic lesions in any other part of the body.

The patient gave a history of repeated exposure to cold and wet, and he himself attributed his illness to this cause. But it is difficult to believe that this alone can have been the cause of the extensive new formation in the membranes, although such exposure is always given as an important cause of meningitis.

As regards the clinical features stiffness and pain in the back, though present, were not so pronounced, and the pains in the limbs, though severe, were not so intense as the pathological changes would lead one to expect. The course of the illness is worthy of note. Pains and weakness of the legs came on very gradually and after reaching a certain stage remained almost stationary or showed very slow increase, but these symptoms, two months before death, grew rapidly worse without apparent cause, and indeed under improved conditions, for he was better cared for in hospital than he had ever been at home; and the subsequent progress of the illness was rapid. A remarkable feature, which I have never seen in so great a degree, was the extraordinary formation of bed-sores during the last weeks of his life. This constituted quite a special feature in the symptomatology. In spite of every care and of the means employed to prevent them, they formed on every bony point on which there was the least pressure, and no doubt greatly hastened his death.

DESCRIPTION OF PHOTOMICROGRAPHS.

FIG. 1 (*Schäfer-Pal Stain*).—Shows cord lying in thickened membranes, which are unstained. The anterior and posterior roots, partly degenerated, can be seen passing out. The circumferential rim of degenerated tissue on periphery of cord (unstained) is well shown and appears even more plainly in fig. 2, where at *A*, outside the nerve-roots, is seen a black mass consisting of fatty substance in a lymphatic space.

In FIG. 2 the extent of the membranes cannot be seen as the celloidin has not been removed.

FIG. 3.—Same stain, more highly magnified, to show exit of anterior root bundles at *B* to form anterior root at *C*, which contains a very large number of degenerated nerve-fibres. At *A* the degenerated margin of cord.

FIG. 4 (*stained in hæmatoxylin and picrofuchsin*); FIG. 5 (*in hæmatoxylin and eosin*).—To show thickened membranes, outer dense layer of thickened dura mater, within this thick layer of looser, very vascular, connective tissue, in which, in fig. 5, vessels and nerve-roots can be seen, and thickened pia mater next to cord. The thickened membranes have become accidentally separated in the anterior aspect in preparing the sections, but were much thinner than on dorsal and lateral surfaces. In fig. 5 part of the cord itself has fallen out in preparing the section.

FIG. 6.—Artery showing marked endarteritis, with some thickening also of adventitial sheath.

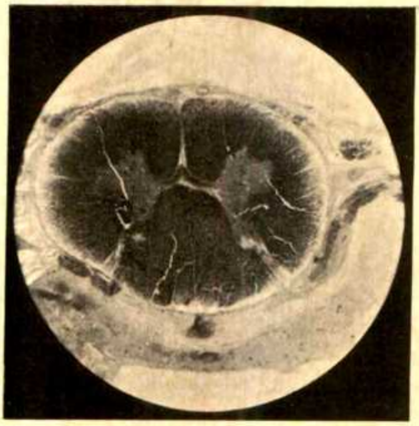

FIG. 1.

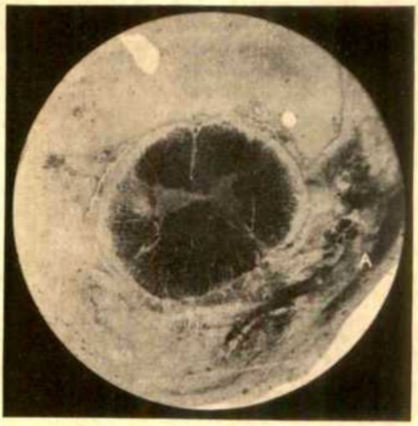

FIG. 2.

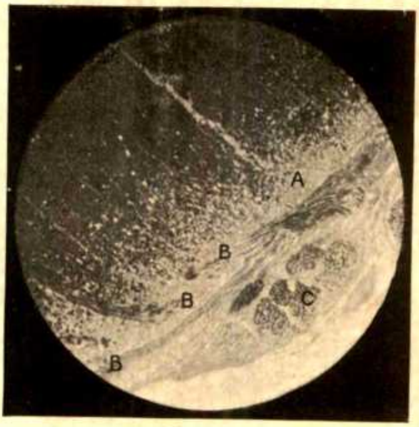

FIG. 3.

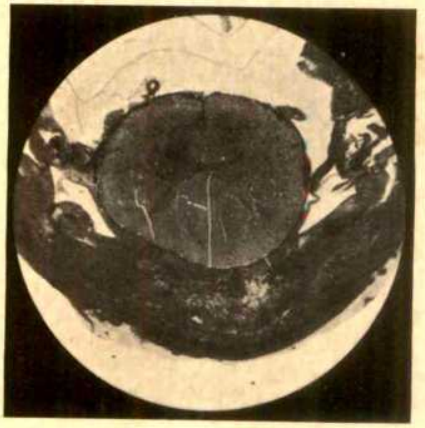

FIG. 4.

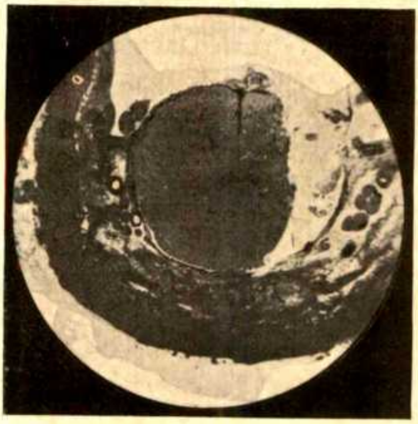

FIG. 5.

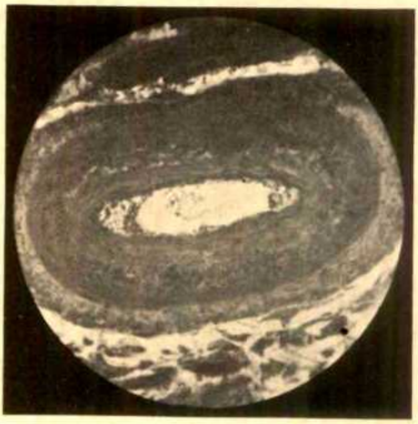

FIG. 6.

DEGENERATIONS IN HEMIPLEGIA: WITH SPECIAL REFERENCE TO A VENTRO-LATERAL PYRAMIDAL TRACT, THE ACCESSORY FILLET AND PICK'S BUNDLE.

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(*From the Museum of the National Hospital.*)

In this paper I propose to describe and discuss certain tracts of fibres which are frequently seen degenerated in the human subject in association with the pyramid in cases of hemiplegia. These tracts include:—(1) A ventro-lateral pyramidal tract; (2) The accessory fillet; (3) Pick's bundle.

My best thanks are due to the Medical Staff of the National Hospital for permission to make use of their cases, and to Professor Victor Horsley and Dr. James Collier for valuable suggestions and criticisms. For the microphotographs I am also indebted to Mr. Horsley.

METHOD.

In each case the whole central nervous system has been first hardened in formalin; the brain for seven days in a 20 per cent. solution, the cord for three days in a 10 per cent. solution. They were then cut into blocks of a convenient size and stained in Busch's fluid (an aqueous solution of osmic acid and sodium iodate) until well penetrated; serial sections were then cut in celloidin and mounted in balsam. Some sections, however, were counter-stained before mounting by Robertson's method in order to demonstrate more clearly the general undegenerated structure.

Sections were stained of all regions from the crura cerebri to the coccygeal region; in one case sections were made through the internal capsule, corona radiata and part of the cerebral cortex.

In this way it was possible to trace the degenerations from their point of origin to their regions of distribution.

It will thus be seen that a modified Marchi method was alone relied upon to determine the degeneration.

Case 1.—*Clinical history.* Andrew H., aged 52, was admitted into the National Hospital, under Dr. James Taylor, on December 4, 1900, thirty-seven days after the sudden onset of right hemiplegia and aphasia in association with old rheumatic endocarditis. There was complete paralysis of the arm and leg, partial paralysis (of the supranuclear type) of the right side of the face, the paralysis being equally marked in voluntary, emotional and associated movements: there was complete motor aphasia and much mental disturbance. The nervous condition remained unaltered until his death, on January 7, 1901, from heart failure.

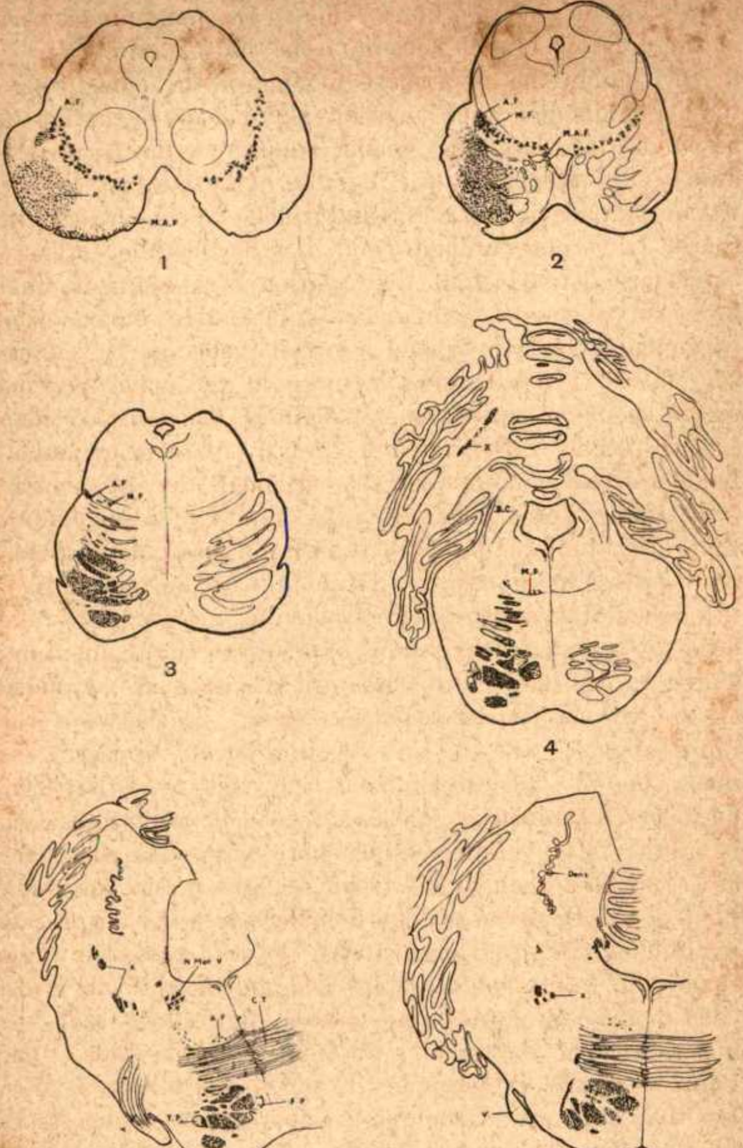
Autopsy.—The mitral valve was found to be almost blocked by large vegetations covered with laminated clot; there were several recent infarcts of the left kidney and the spleen.

There appeared to have been blocking of one of the main branches of the left middle cerebral artery in the Sylvian fissure. The resulting softened area seen on the surface of the cortex was small and included only Broca's area, a small piece of the anterior end of the upper temporal convolution and the lower end of the ascending frontal convolution; the whole of the island of Reil appeared to be softened. Coronal sections through the centre of the insula showed that the lesion involved the deeper structures down to the optic thalamus, the upper and outer edge of which was alone softened, the red nucleus being intact. The lenticular and caudate nuclei were largely softened, but the internal capsule only partly involved with the original lesion, the frontal and temporal fibres of the capsule escaping injury; the whole of the fibres from the Rolandic area were interrupted in this region.

Degenerations.—Coronal sections of the left cerebrum through the island of Reil confirmed the extent of the degeneration as inferred from macroscopic examination, only the upper one-fourth of the optic thalamus (exclusive of the red nucleus) being involved. Consequent upon this lesion there was extensive degeneration in the left crus cerebri, but none in the right crus, nor in the tegmental region of either side. The pyramidal fibres in the middle region of the crusta were all degenerated; the inner two-fifths (the fronto-pontine region) and the outer one-sixth (the temporo-pontine region) of the longitudinal fibres were in the main free from disease, but the distinction between degenerated and undegenerated regions was not sharp, the two sets of fibres intermingling at the junction. One small degenerated bundle, somewhat less than one-twelfth the size of the pyramid in this region, and lying immediately dorso-lateral to the latter, gradually becomes more dorsal and lateral, until at the level of the posterior corpus quadrigeminum (case 1, fig. 2) its fibres mingle with the outermost cells of the substantia nigra, and the degenerated tract lies close up against the fillet. Lower sections show that this tract spreads itself out in the uppermost part of the pons, and that its fibres come to lie in small groups in the middle fillet itself (case 1, fig. 3). At first they occupy the outer part of the fillet on its ventral aspect, but they quickly pass further towards the middle line and gradually assume a more dorsal aspect, many of them leaving the fillet and being distributed to the tegmentum as single fibres. It was impossible to trace individual fibres to the nuclei, but in the main they appeared to end in the region of the motor nucleus of the trigeminus and the facial nucleus of the same side; a few fibres crossed the middle line and were distributed to the corresponding nuclei of the opposite side. No degenerated fibres were visible in the tegmentum of the lower part of the pons below the level of the facial nucleus, nor could any be seen in the inter-olivary region of the medulla.

Through the upper part of the pons certain reinforcements to this tract—the "accessory fillet"—take place

CASE 1.



The accessory fillet (*A. F.*) and mesial accessory fillet (*M. A. F.*) separate from the pyramid in 1, and pass to the middle fillet (*M. F.*) in 3. In 5 accessory fillet fibres pass to the motor nucleus of trigeminus (*N. Mot. V.*) *X* represents the bundle described on p. 471.

from other degenerated fibres lying in the most dorsal part of the crusta. It will be seen from the diagram (case 1, fig. 3) that in the uppermost regions of the pons, where the pyramidal and other longitudinal fibres are beginning to be cut up into larger or smaller bundles by the transverse fibres, that certain of the bundles lying nearest to the tegmentum are narrow horizontal bands of a concavo-convex shape, lying near to and parallel with the fillet. These bands are derived from that portion of the longitudinal fibres of the crus which lie immediately outside the pyramidal fibres proper, and between them and the accessory fillet. These bundles, as seen in the upper pontine region, contain many degenerated fibres, but none of them are as heavily degenerated as are the larger pyramidal bundles situated more ventrally, although they show more degeneration proportionately to the number of healthy fibres than does the middle fillet. It is from these bundles that fibre accessions are constantly coming to the middle fillet throughout the upper pontine region as the accessory fillet becomes attenuated by sending fibres into the tegmentum. These narrow bundles of fibres are still seen as low down as the corpus trapezoides (case 1, fig. 6), between the fibres of which they lie in very thin sheets, appearing as mere lines in cross section; but below this level they have disappeared completely and the only degeneration to be seen is in the left pyramid. Other fibre accessions reach the accessory fillet from Spitzka's bundle (the mesial accessory fillet of von Becterew). This bundle leaves the degenerate pyramid in the crus (case 1, fig. 1) and passes inwards round the fronto-pontine tract and among the innermost cells of the substantia nigra (case 1, fig. 2) to reach the mesial end of the middle fillet of the same side. The number of fibres thus supplied to the middle fillet is small—less than a quarter of the number supplied by the accessory fillet. At about the level of the decussation of the fourth nerves the two sets of fibres mingle and can no longer be distinguished.

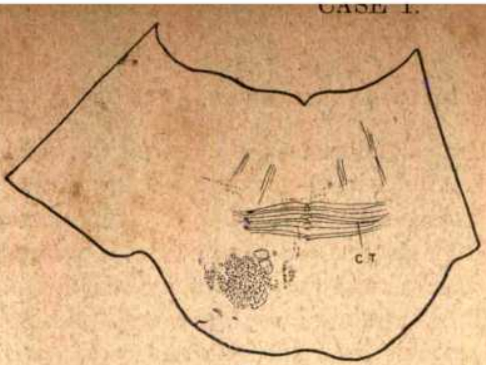
The behaviour of the pyramid in the pons calls for some comment. When the descending fibres from the crus are split up first into separate bundles by the crossing fibres of the pons, it is noticed at once that the bundles are not of equal size, but that in the main those bundles which are ventrally placed are the larger, and that there is a progressive narrowing in the vertical direction of the bundles as they near the tegmentum. In this case the fronto-pontine and temporo-pontine tracts are undegenerated and can, therefore, be fairly easily traced to their region of distribution. It will be seen from the diagrams (case 1, fig. 3) that the fronto-pontine fibres occupy the innermost of the bundles described, or in the case of larger bundles, their most mesial parts. These undegenerated fibres are seen to rapidly diminish in numbers through the upper and middle pontine region, and have quite disappeared at the level of the exit of the seventh nerve.

The temporal and occipital fibres—occupying the outer parts of the bundles—disappear at a slightly higher level; in both cases the loss of fibres is quite gradual, and they appear to become lost amongst the nuclei of the pons.

Sections through the lowest limits of the pons show that practically all the pyramidal fibres on the left side, and none on the right side, are degenerated. In the lowest pontine region the fibres apparently undergo considerable re-arrangement; the outer bundles especially changing their position, so that in the main those which lay dorso-lateral come to lie ventro-lateral, forming the lateral apex of the triangular pyramid as it is formed in the medulla. At first the medullary pyramid forms a triangle flattened dorso-ventrally, but it gradually becomes compressed laterally so that just before the decussation its shape is almost rectangular (case 1, fig. 12, and microphotograph), with one of the shorter sides adjacent to the ventral surface of the medulla.

Ventro-lateral pyramidal tract.—At this point a compact bundle of heavily degenerate fibres—some large, some small—is seen to become separated from the main mass. Passing rapidly outwards along the ventral edge of the medulla, it assumes a lenticular shape in the ventro-lateral region of the cord and maintains this position as far as it can be traced. From its mode of origin here, and from the position

CASE I.



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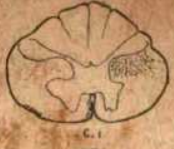

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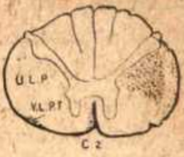

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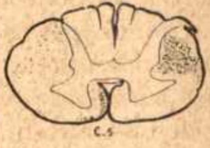

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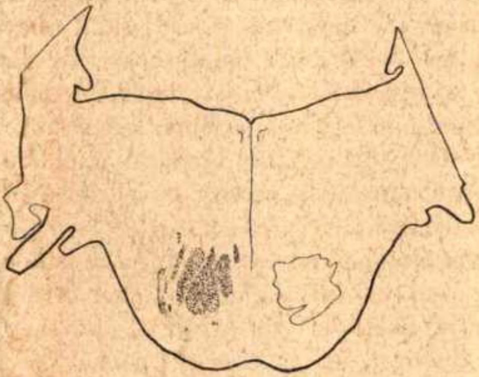

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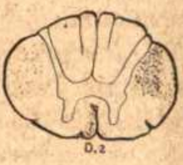

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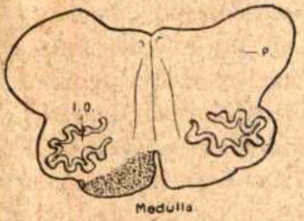

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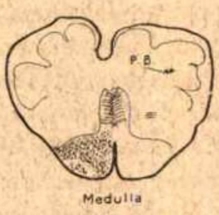

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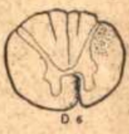

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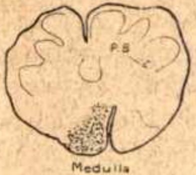

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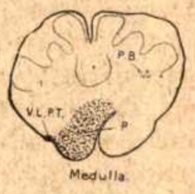

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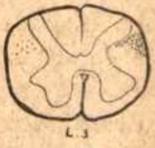

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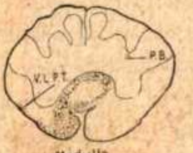

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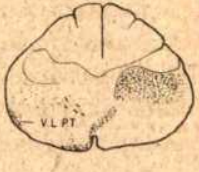

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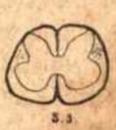

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The ventro-lateral pyramidal tract (*V. L. P. T.*), and Pick's bundle (*P. B.*), are both degenerated.

it assumes in the spinal cord, I propose for this tract to use the term "ventro-lateral pyramidal tract." In this case it could be traced as a distinct tract as far as the third cervical segment of the cord, below which the fibres become scattered, but still lie in the ventro-lateral marginal region; they could not be traced beyond the upper dorsal segments.

The pyramid, after giving off this tract, decussates with its fellow, and at the same time gives off from its left side a bundle of fibres which does not cross the middle line but curves sharply backward and at once spreads out, mingling with the undegenerated fibres of the right pyramid. These are the lateral uncrossed fibres of the pyramid.

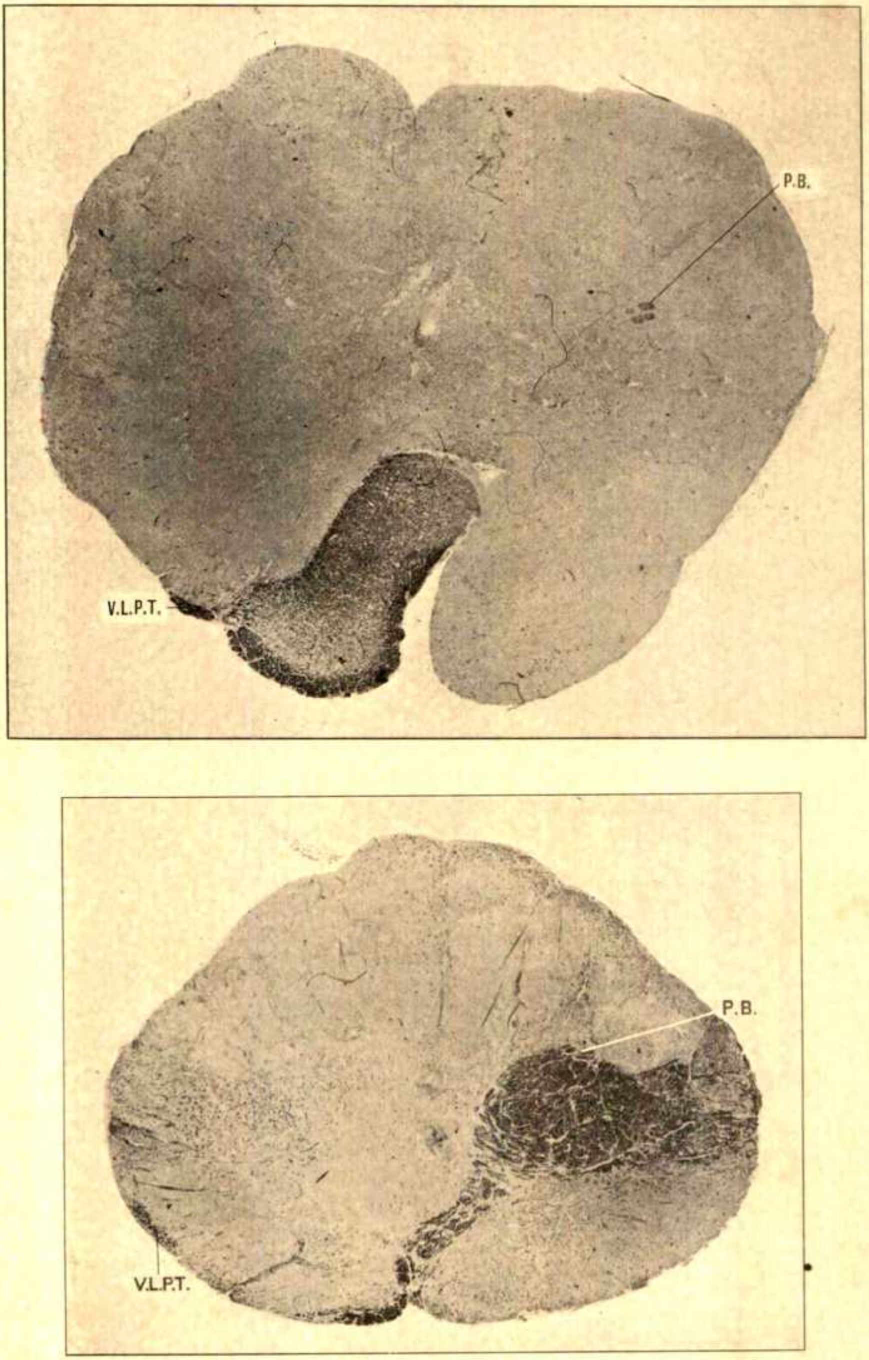
At the lower end of the decussation there is left behind an uncrossed tract about one-eighth as large as the crossed pyramid lying near the anterior fissure—the direct pyramidal tract. No fibres could be seen going to the opposite direct pyramidal region (ventral crossed bundle), nor to the opposite ventro-lateral region.

The crossed pyramid quickly assumes an equilateral triangular appearance, one side being limited by the posterior horn, a second by a slightly curved line parallel to the outer edge of the cord, and the third by a line drawn outwards and slightly ventralwards from the region of Clarke's column. The direct cerebellar tract thus separates it in the main from the edge of the cord, but many fibres are seen to lie separated from the main mass, some mixed with the fibres of the direct cerebellar tract, and more at the periphery of the cord both outside and ventral to that tract.

The crossed pyramidal tract can be traced down to the coccygeal region. The homolateral fibres can be traced down to the fifth sacral, and the direct pyramidal tract to the first lumbar segments of the cord.

Pick's bundle.—Another bundle of fibres needs description. In the lower sections of the medulla are seen degenerated four small bundles of fibres closely massed together at the inner side of the substantia gelatinosa. Traced upwards these fibres—which correspond to what is usually known as Pick's bundle—become rapidly diffused and lost in the

CASE 1.



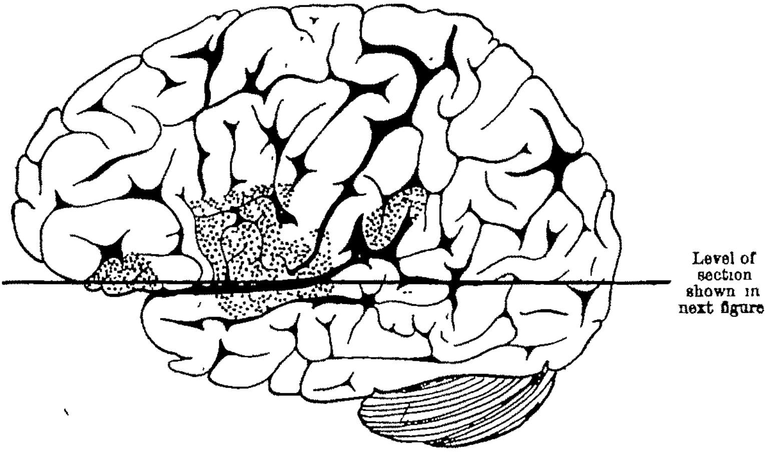
Microphotographs of sections of medulla, showing the origin of the ventro-lateral pyramidal tract and Pick's bundle.

region of the nucleus ambiguus, not a single fibre being visible in this region at the lowest pontine level, and none of them can be seen to cross the middle line. Traced downwards towards the pyramidal decussation these fibres retain their position but increase in number and in compactness, lying close to the ventro-lateral aspect of the posterior horn at its junction with the substantia gelatinosa in the upper part of the first cervical segment. Here the degenerated bundle appears to gradually merge into the degenerated crossed pyramid which now lies immediately adjacent to it. For several sections, however, after the crossing fibres of the pyramid have reached them, the bundles remain distinct and are easily distinguished. Many of the fibres then take an oblique course and are seen as though cut almost longitudinally in transverse sections, giving the impression of fibres which are curving rather sharply at this point. It was quite impossible to trace any of the fibres to the opposite (left) side through the decussation of the pyramids, as they lose their bundle form and are no longer distinguishable from the rest of the degenerated pyramid.

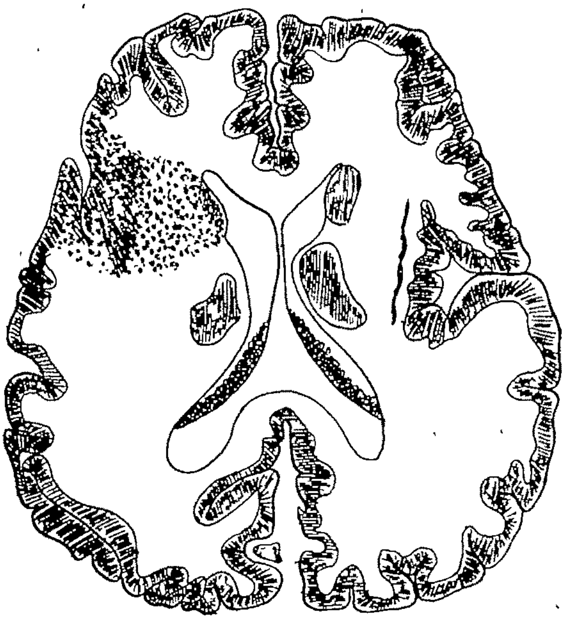
Besides the tracts already described, which all appear to have been the result of the cerebral lesion, another set of fibres having its origin in the lateral pontine region at the lowest level of exit of the fifth nerve was markedly degenerated (case 1, fig. 5). The compactness of the fibres strongly suggests that they might be nerve-roots, but their position makes such an assumption difficult to accept. There may have been a small separate focus of softening—possibly due to another embolus—but the area involved is very small and no lesion was seen: there was no downward degeneration from this point, nor could any connection with the pyramidal or other degeneration be made out, although every section was examined. The four or five heavily degenerated bundles, close together at their commencement and lying just dorso-lateral to the superior olive pass slightly upwards and rapidly curve outward and dorsalwards, diverging as they ascend in the middle peduncle of the cerebellum. A few fibres, split off from the rest, pass on the inner side of the dentate nucleus and are lost—some in the white matter over the lateral aspect of the fourth ventricle, some in the nuclei tectis, and a few in the dentate nucleus itself. The main mass passing dorsalwards courses outside the dentate nucleus and then curves sharply to pass in a ventro-lateral direction into the peduncle of the flocculus of its own side (case 1, fig. 4). None of the fibres, except a few passing to the vermis, could be seen crossing to the opposite side and none degenerated downwards.

I do not know what this tract represents. Its appearance does not suggest that the fibres are merely bundles of the middle peduncle. It is possible that they are part of the direct sensory cerebellar tract of Edinger, passing from the auditory nerve to the flocculus; they certainly have no connection with the pyramidal degenerations.

Case 2.—Clinical history. Annie W. was admitted to the National Hospital, Queen Square, under Dr. Hughlings Jackson, on April 15, 1901, suffering from right hemiplegia and aphasia. She had been the subject of heart disease since infancy, and ten years before admission had suffered from epilepsy, which ceased entirely under treatment in about two years. For a year before admission attacks of œdema of legs had come on, clearing up when she lay in bed. On February 7, 1901, after being in her usual health, she woke up with inability to use the right arm or leg, and loss of speech; the leg had slowly improved, but the arm and speech remained stationary until admission. When seen on April 15 she was suffering severely from heart failure, there being evident signs of mitral stenosis, aortic regurgitation and tricuspid regurgitation; the liver was strongly pulsating. There was no hemianopia or hemianæsthesia. Motor aphasia was complete; she understood spoken words, could understand simple written sentences, could copy with the left hand, but could not write from dictation. The right pupil was larger than the left, both reacting well to light and on accommodation. The lower facial segment on the right side was almost completely paralysed—equally for voluntary and emotional movements. No difference could be detected between the power of the masseter and temporal muscles. The tongue deviated slightly to the right. There was almost complete flaccid palsy of the right arm—all movements below the elbow being completely lost—and a considerable paresis of the right leg. All the tendon-jerks were increased in the paralysed arm and leg, and the right plantar reflex was of the extensor type, the left being flexor.



1.—Surface of brain: softened areas are dotted.



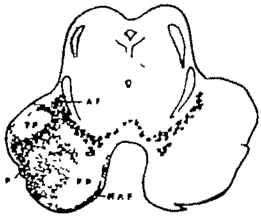
2.—Horizontal section of brain at level shown in figure 1.

She was kept in bed and given alcohol and digitalis, and in a few days the heart failure was largely overcome. The nervous condition, however, did not alter until May 14, when she became suddenly unconscious with stertorous breathing and slight general convulsions; she never recovered consciousness, but died next day.

AUTOPSY.—There was old disease of the mitral, aortic and tricuspid valves, and the usual associated visceral changes. The brain (case 2, fig. 1) showed areas of softening around the Sylvian fissure, involving mainly the island of Reil, the first temporal, the lower part of the ascending frontal and parts of the inferior frontal and angular convolutions. Several small branches of the Sylvian artery were occluded, but the main artery and some of its branches were patent. A horizontal section of the brain through the centre of the fissure of Sylvius (case 2, fig. 2) showed that the softening included the whole of the island of Reil and subjacent white matter down to the lateral ventricle, the head of the caudate nucleus being involved. A series of sections parallel with the first showed that the softening also included the greater part of the lenticular nucleus, but that nowhere was the optic thalamus encroached upon; this was also confirmed microscopically. The anterior limb and a few of the most anterior fibres of the posterior limb of the internal capsule were also destroyed. On the right side the middle cerebral artery was completely blocked by red gelatinous clot, and the island of Reil and neighbouring convolutions were red and slightly soft; it was evident that an embolism had occurred on this side and had been the immediate cause of death. No degeneration resulted from this lesion, which was probably not more than thirty-six hours old.

THE DEGENERATIONS.—In this case many of the *fronto-pontine* fibres were degenerated, being probably cut off from their cell-origin by the lesion in the internal capsule. Some escaped injury, so that in the upper pontine region many undegenerated fibres are seen on the inner side of the pyramid. As in case 1 the undegenerated fibres disappeared gradually and none could be seen at the level of exit of the seventh nerve.

CASE 2.



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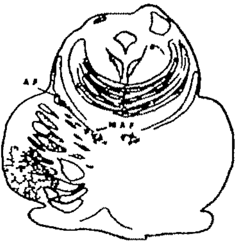

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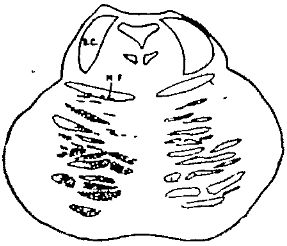

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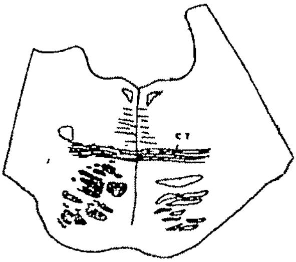

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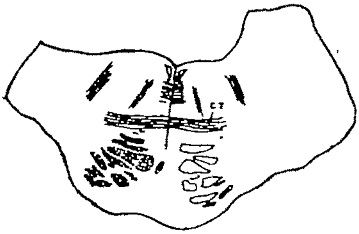

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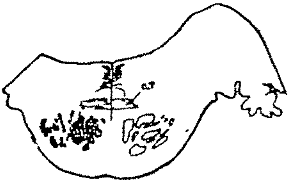

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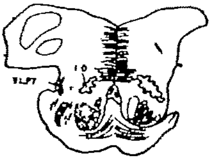

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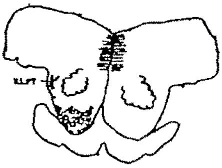

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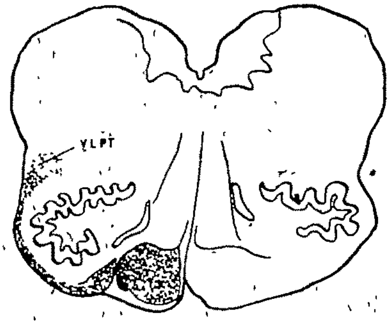
Figs. 4—8 show the origin and course of the accessory fillet (*A. F.*) and the mesial accessory fillet (*M. A. F.*). Figs. 10, 11 show the origin of the ventro-lateral pyramidal tract (*V. L. P. T.*).

CASE 2.



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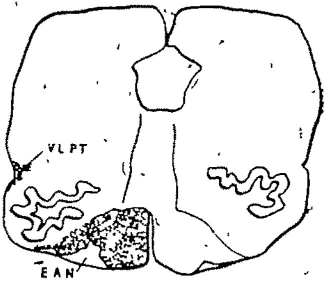

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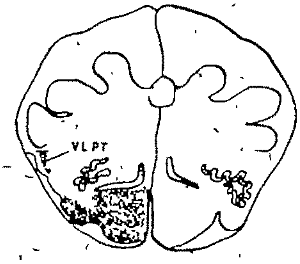

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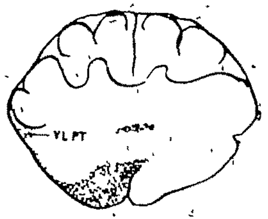

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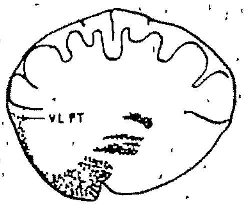

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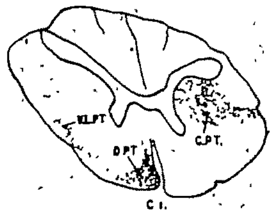

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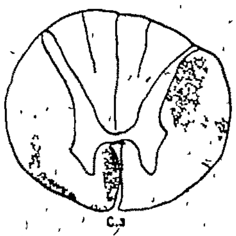

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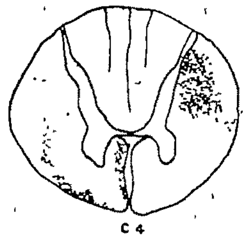


19

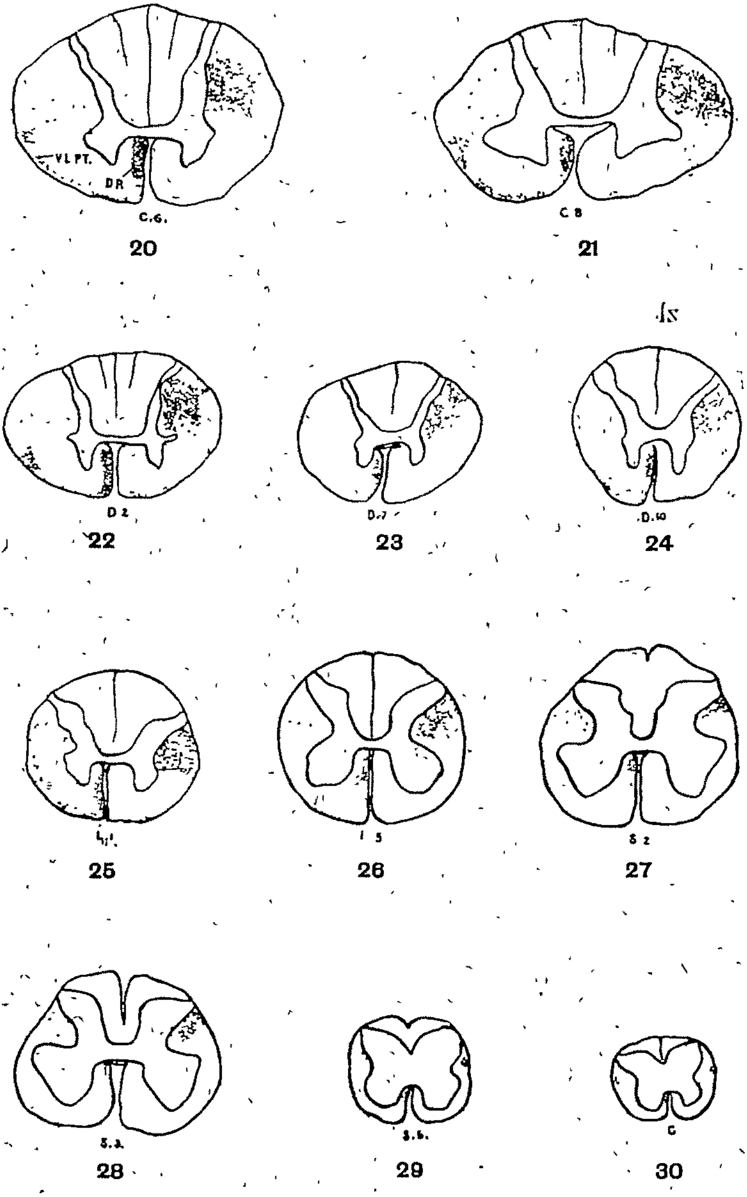
Figs. 12—16 show the arrangement of the pyramid and the ventro-lateral pyramidal tract at various levels of the medulla. Figs. 17—19 show the position of the large ventro-lateral tract in the cord.

The ansa lenticularis was degenerated on the left side. Its fibres could be traced from the softened lenticular nucleus curving below the internal capsule and upwards to the optic thalamus of the same side. Many fibres, presumably belonging to this system, passed transversely through the internal capsule from the lenticular nucleus; most of them ended in the corpus Luysii, but a few passed through or round that body to the optic thalamus proper. The optic tract, the bundle of Vicq d'Azyr and the fasciculus retroflexus of Meynert were not degenerated. Spitzka's bundle formed a loop of degenerated fibres passing from the pyramid in the crus inwards towards the mesial aspect of the middle fillet.

The *pyramidal degeneration* differed only in minor details from that described in case 1 in the internal capsule, mesencephalon and upper half of the pons Varolii. The *accessory fillet* was given off in exactly the same way (case 2, figs. 4 and 5), so that the description in case 1 represents its course with sufficient accuracy. It received many accessions from the thin layers of pyramidal fibres lying subjacent to the middle fillet, and was in the main distributed to the motor nuclei of the fifth and the facial nuclei, chiefly those of the same side, partly those of the opposite side. Many fibres to the opposite nuclei could be seen splitting off from the innermost bundles of the pyramid, and passing up the raphe crossed to the opposite side and turned horizontally outwards on reaching the tegmentum. Many pyramidal fibres were in this way distributed also to the hypoglossal nuclei—chiefly of the opposite but partly of the same side; but no accessory fillet fibre could be traced as low down as this level. There was again no degeneration in the interolivary region beyond the fibres distributed by the pyramid to the medullary nuclei.

Below the corpus trapezoides the appearance of the pyramids differs considerably from that seen in case 1. The bundles in case 2 are smaller and more scattered, and in the lowest sections of the pons (case 2, fig. 10) one bundle is seen to be lying in a dorso-lateral position apart from the other bundles, and on a horizontal level with the upper end

CASE 2.



20 21 22 23 24 25 26 27 28 29 30

Figs. 20—30 show distribution of ventro-lateral pyramidal tract (*V. L. P. T.*).

of the inferior olive. This bundle, the ventro-lateral pyramidal tract, continues to remain distinct from the rest of the degeneration, but certain fibres, cut longitudinally in cross sections, are seen to be passing between the two, forming a connecting link around the inferior olive (case 2, fig. 12).

The majority of these fibres now form a bundle which corresponds closely in position with Gowers' tract, and this association they retain almost to their distribution. At the decussation of the pyramids a large direct pyramid is left behind, whilst the ventro-lateral pyramidal fibres spread out on the periphery of the cord occupying the whole region from the direct pyramidal to the direct cerebellar tracts (case 2, fig. 18). It will be noticed, however, that many of the more dorso-lateral of the fibres are not placed at the periphery of the cord, but lie somewhat deeper, the degeneration here fading off into the slightly degenerated crossed pyramid of the left side.

The lateral uncrossed fibres of the crossed pyramid are about the same in number as in case 1, and arise from the decussating pyramid as a distinct bundle. There are a few degenerated fibres in the ventral pyramid of the opposite (right) side, and along the edge of the cord in the ventro-lateral region.

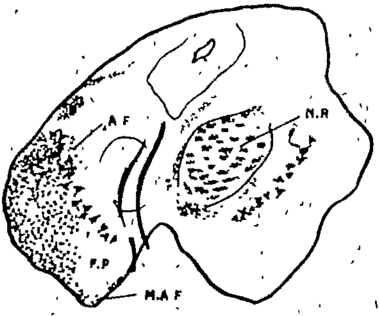
The crossed pyramidal tract and lateral uncrossed fibres could be traced to the coccygeal region; the direct pyramidal, as a definite bundle, as low as the third sacral, and the ventro-lateral tract as low as the fifth lumbar.

Pick's bundle was not degenerated.

Case 3.—*Clinical history.* Edgar W. was admitted to the National Hospital, on April 17, 1900, with symptoms of cerebral tumour of about five months' duration. On April 28, 1900, he was trephined in the left temporo-parietal region, a piece of bone about 3 inches square being removed; the dura was opened and the brain—which was evidently under great pressure—bulged considerably. The scalp was sutured over it. The wound healed well.

After the operation the cerebral hernia gradually increased in size until, at the time of his death on January 8, 1901, the hernia formed a mass almost as large as the head. There was complete hemiplegia and aphasia for six months before death, whilst since October, 1900, consciousness had become progressively less, deepening into coma for the last three weeks.

CASE 3.



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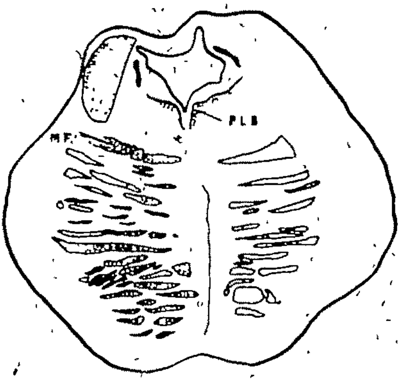

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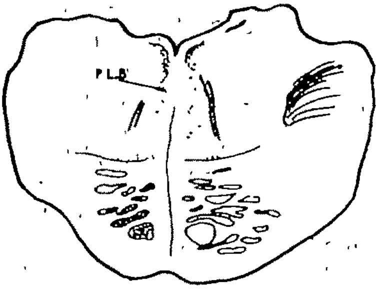

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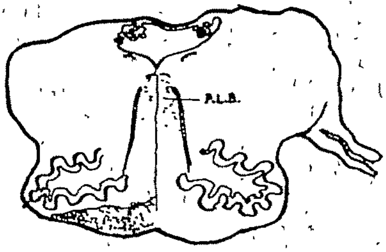

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Fig. 1 shows degeneration of the accessory fillet (*A. F.*) and the mesial accessory fillet (*M. A. F.*). Both posterior longitudinal bundles (*P. L. B.*) are degenerated.

AUTOPSY.—The hernia was a carcinoma which had involved almost the whole of the left cerebral hemisphere and basal ganglia, the corresponding cerebral peduncle being much elongated and thinned.

A small portion of the occipital lobe and the most anterior of the frontal convolutions were intact; the island of Reil, lenticular nucleus, internal capsule (genu and posterior limb), caudate nucleus and upper part of the optic thalamus were almost completely destroyed by the growth. The most anterior of the fibres of the anterior limb of the internal capsule were spared. No other gross lesion was found in the nervous system.

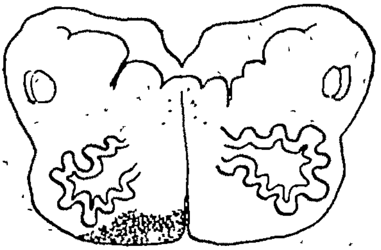
THE DEGENERATIONS.—Owing to the degeneration being of long standing, the Marchi staining is not so clear and brilliant as in cases 1 and 2, and it is impossible in the mid-brain and pons to follow out individual fibres or small tracts: in the cord and medulla the degenerations stained well.

Sections through the internal capsule show that the only fibres free from degeneration are the most anterior of the frontal ones. A mass of degenerated fibres passes to the opposite optic thalamus, in the red nucleus of which they form fine bundles of degeneration as well as a sort of fleece on its inner side (case 3, fig. 1). A few fibres also pass to the red nucleus of the same side.

Another bundle of fibres is seen passing from the outermost part of the capsule towards the corpus quadrigeminum anterius of the same side. The posterior longitudinal bundle of the opposite side is also partly degenerated. A few degenerate fibres are seen in the corresponding bundle of the left side, and both these sets of fibres can be traced through the pons to the medulla oblongata, when they become diffuse and are lost in the interolivary layer.

The accessory fillet.—As in cases 1 and 2 some of the fibres of the internal capsule lying immediately outside the pyramid become detached from the rest of the degenerated bundles, and at the uppermost level of the pons lie in a compact bundle at the junction of the crusta and the tegmentum, near the periphery; later they pass inwards and become diffused among the fibres of the middle fillet; some are seen to cross the middle line and for a time lie in the opposite middle fillet, but their further destination cannot be traced. Spitzka's bundle was also degenerated

CASE 3.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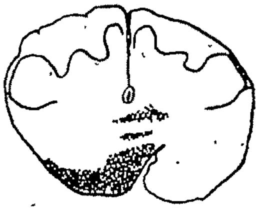
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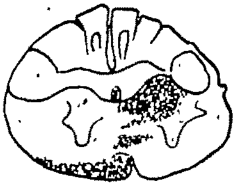
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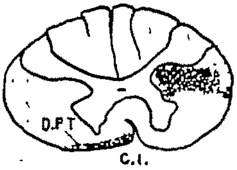

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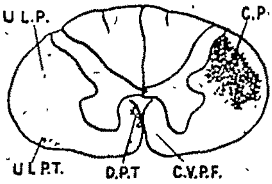

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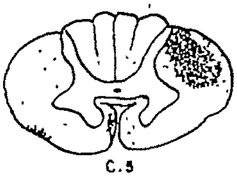

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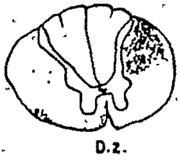

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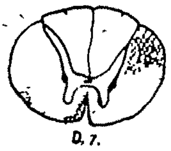

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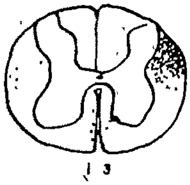

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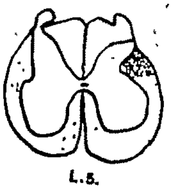

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The ventro-lateral pyramidal tract in this case arises from the direct pyramidal tract (*D. P. T.*) at the level of the first cervical segment (figs. 8 and 9).

in this case, and took a similar course to that described in case 1.

In the upper part of the medulla the left pyramid is wholly degenerated, many of the fibres being represented by vacuoles and the rest showing as black dots. There is some scattered degeneration in the interolivary region, partly derived from the diffusing fibres of the posterior longitudinal bundles, and perhaps partly from the descending fillet degeneration.

The pyramid in the medulla remains of a more or less triangular shape until it begins to decussate with its fellow. The crossed pyramid is then formed in the usual way, and after the homolateral fibres have been given off a mass of fibres is left behind lining adjacent portions of the mesial and ventral aspects of the left side of the medulla. This mass of fibres retains this L-shaped form until it reaches the first cervical segment, when it divides into two portions; the larger—the direct pyramid—remains lining the mesial aspect of the ventral fissure, whilst the smaller (about one-half to one-third the size of the former) passes sharply outwards along the edge of the cord to reach the ventro-lateral region. A little lower down these fibres (which evidently correspond to what in cases 1 and 2 was called the ventro-lateral pyramidal tract) assume a triangular shape with the base at the periphery of the cord and the apex pointing towards the central canal. The tract is here massed around a deeply-marked fissure—the most evident fissure, in fact, in the ventro-lateral region of the cord, and they do not form a pure degenerated tract, but are situated amongst a rather larger number of healthy fibres (case 3, fig. 9). They can readily be traced downwards as forming a well marked tract as low as the fifth dorsal segment, and always preserving a more or less flattened triangular shape in the same region of the cord. Below this level the fibres become scattered, but individual fibres are seen down to the lumbar region. In its course the tract is seen to consist of both coarse and fine fibres, the fine predominating.

The direct pyramidal tract ends at the second lumbar segment. The crossed pyramidal tract can be traced to

CASE 4.

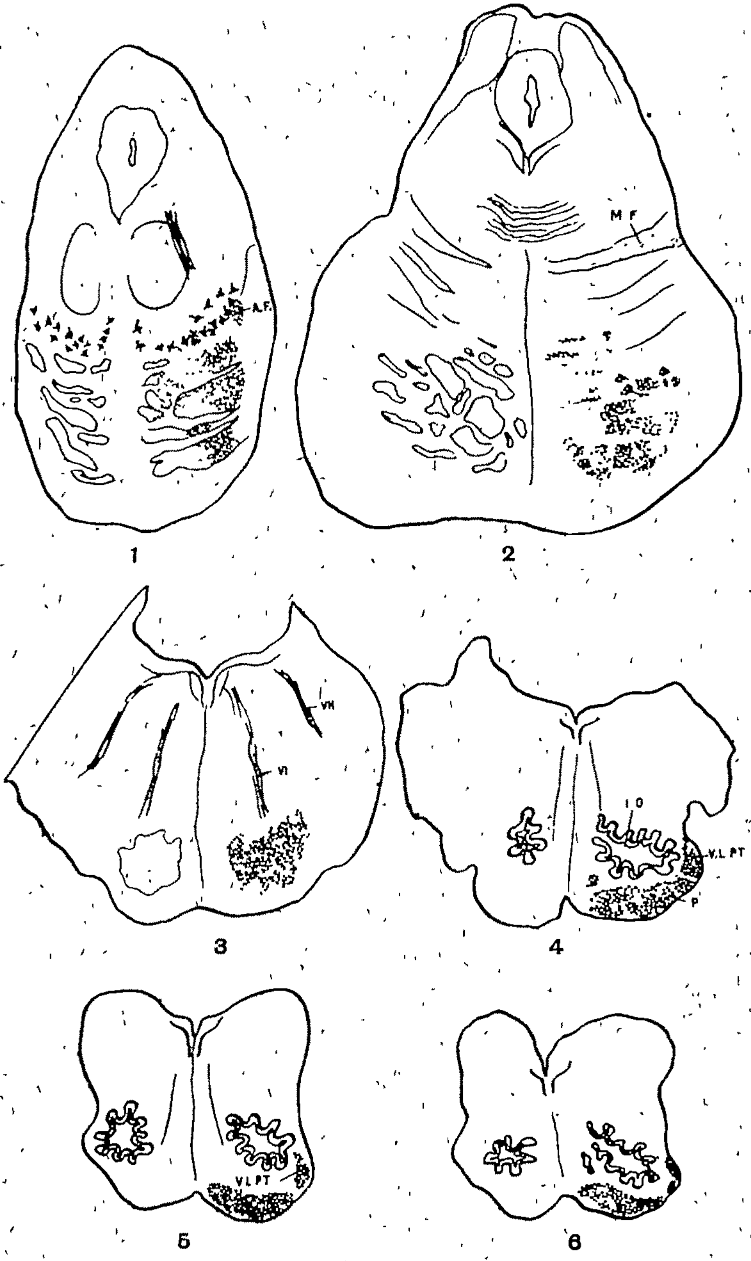


Fig. 1 shows the origin of the accessory fillet (*A. F.*), and fig. 2 its diffusion among the fibres of the middle fillet (*M. F.*). The ventro-lateral pyramidal tract (*V. L. P. T.*) arises in the upper part of the medulla (fig. 4).

the lowest sacral region, and the homolateral fibres of the crossed tract can be traced almost as far.

There were no fibres in the opposite direct pyramid. A considerable amount of posterior column degeneration—apparently due to certain posterior roots—is not figured in the diagram; none of this could be traced above the gracile and cuneate nuclei.

Case 4.—*Clinical history.* John R. was admitted to the National Hospital, under Dr. Bastian, on July 4, 1900, with signs and symptoms suggesting a focal lesion in the right motor area. He was trephined in the right fronto-parietal region on September 5, but no lesion was then found.

He gradually became more and more hemiplegic, but up to the time of his death preserved to a considerable extent all movements of face, arm and leg. Death occurred suddenly on January 5, 1901, during a convulsion.

AUTOPSY.—A large chronic abscess was found in the right fronto-parietal region. At the region of the trephine hole the abscess wall was attached to the dura mater, but elsewhere the abscess was buried in the cerebrum. The deepest part of the abscess reached the level of the corpus callosum; the basal ganglia on both sides being macroscopically intact.

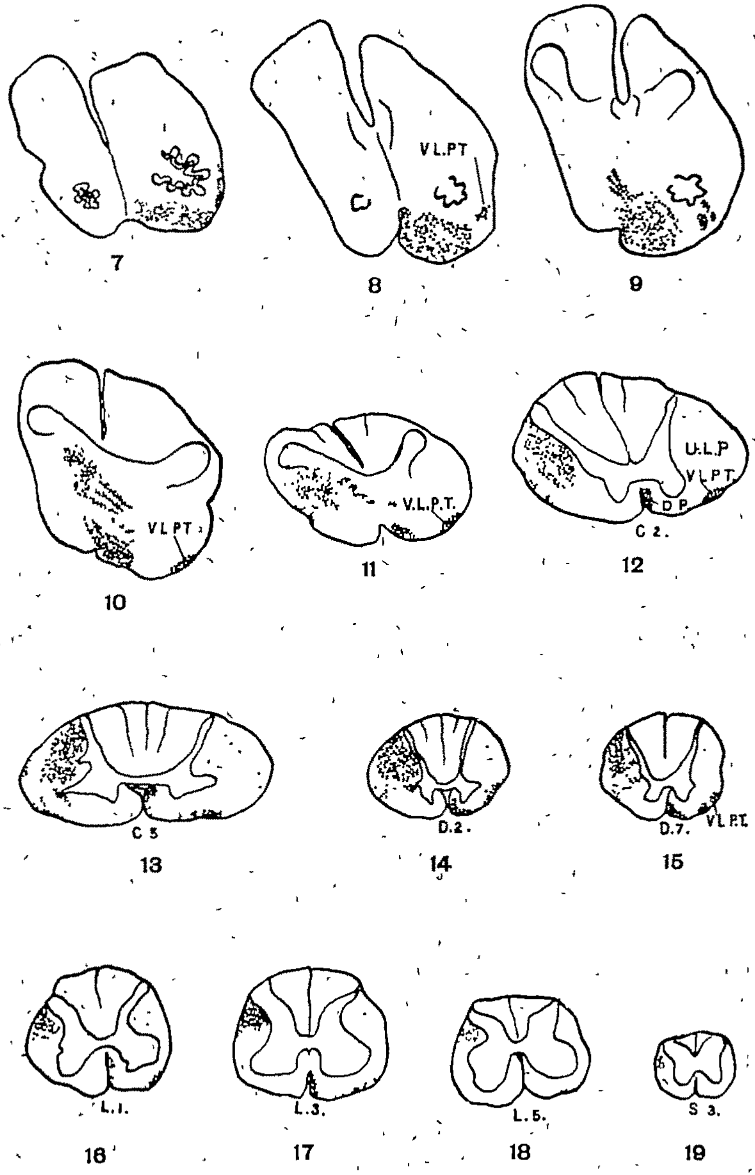
No other gross lesion of the nervous system could be made out.

THE DEGENERATIONS.—In this case the resulting degenerated fibres did not stain so clearly as in cases 1 and 2, so that in the pons individual fibres could not easily be traced. Microscopical examination confirmed the view that the optic thalamus was not involved in the lesion, but parts of the caudate and lenticular nuclei, and much of the internal capsule, were destroyed.

There was a heavy degeneration of the pyramidal fibres in the right crus cerebri, and some, but probably not many, of the frontal fibres were affected. In the main the temporal fibres were intact.

The behaviour of the pyramid in the pons corresponds fairly closely to that observed in cases 1 and 2, and needs no special description.

CASE 4.



Figs. 7—19 show the distribution of the ventro-lateral pyramidal tract (*V. L. P. T.*).

The *accessory fillet* begins to separate from the pyramid at the lowest level of the mesencephalon, and forms a well-marked bundle of fibres which, at the upper part of the pons, partly mingles with the outermost cells of the substantia nigra. Lower down the fibres quickly mingle with those of the middle fillet, forming at first a diffuse degeneration in its outer two-thirds; the fibres then become aggregated into small bundles, and are seen as such in sections at the level of exit of the fifth nerve. They pass into the tegmentum, but the staining was not sufficiently clear to trace any fibres to their nuclei of distribution. The accessions to these fibres from the more dorsal of the pyramidal bundles of the pons are not so marked as in cases 1 and 2.

Pick's bundle was not degenerated. The *ventro-lateral pyramidal* tract is given off at the lower part of the pons varolii. The pyramid here forms a single compact bundle of degenerated fibres. The most dorso-lateral of the fibres quickly becomes separated by a slight interval from the other fibres (case 4, fig. 4), so that when the inferior olive appears, the ventro-lateral pyramidal tract forms a triangular bundle of degeneration, covering the outer surface of the olive not quite in contact with the apex of the triangle formed by the pyramid proper. This position the ventro-lateral tract maintains throughout the medulla oblongata, varying in shape but never re-uniting with the pyramid. In the upper part of the cervical region of the spinal cord (case 4, fig. 11) it forms a lenticular mass at the periphery which can be traced as a definite tract as low as the third lumbar segment, a few fibres being visible in the sacral region. Most of the fibres seem to be given off at the cervical and lumbar enlargements, but some diminution occurs throughout the dorsal region.

At the decussation of the pyramid, a rather large direct tract is left behind, and the lateral uncrossed fibres arise as a well defined bundle. The direct tract can be traced as a definite bundle visible to the naked eye as low as the first sacral segment.

The shape of the crossed pyramid was in the main similar to that described in cases 1 and 2, but the clump of fibres at the periphery in the ventro-lateral region is here more distinct and comes further towards the ventral fissure. It lies, however, further outwards than the uncrossed ventro-lateral tract and has also a different shape; nor can it be traced as far down, ceasing about the second dorsal region. The crossed tract can be traced down to the coccygeal region.

Case 5—*Clinical history.* George D., aged 57, was admitted to the National Hospital on June 19, 1901, under Dr. Ferrier Three weeks before admission there was a sudden onset of weakness in the right hand; within six hours the right upper and lower extremities were paralysed and speech was a little awkward, and twelve hours later he could not speak or use the right arm or leg.

On admission he was quite helpless and in a generally weak condition; mentally he was dull and drowsy, but appeared to understand simple sentences. The right arm and leg were completely paralysed, and the right side of the face (in the lower segment) was equally paralysed for voluntary and emotional movements; no face movement on either side could be obtained in association with a strong grip, &c. The tongue deviated markedly to the right. Hemianæsthesia and hemianopia were well marked. He gradually became weaker and died comatose on June 27, no appreciable change having occurred in the motor condition.

AUTOPSY.—There was extensive vascular disease, especially in the arteries of the circle of Willis. The left middle cerebral artery was blocked by a cartilage-like clot, and there was extensive softening in the left hemisphere. Superficially only a circular patch on the left upper frontal convolution (about an inch in diameter) was softened; but sections showed that much of the hemisphere was softened, the caudate and lenticular nuclei being almost wholly and the upper part of the optic thalamus partly involved. The lesion did not extend into the red nucleus. Of the internal capsule, only part of the anterior genu remained intact.

THE DEGENERATIONS.—The thalamic and hypothalamic regions were not stained. In the upper pontine region there was well-marked degeneration of the pyramidal and temporal fibres, whilst the *accessory fillet*, pursuing a course very similar to that described in case 1, caused a very heavy degeneration to appear in the middle fillet, so that at the

CASE 5.

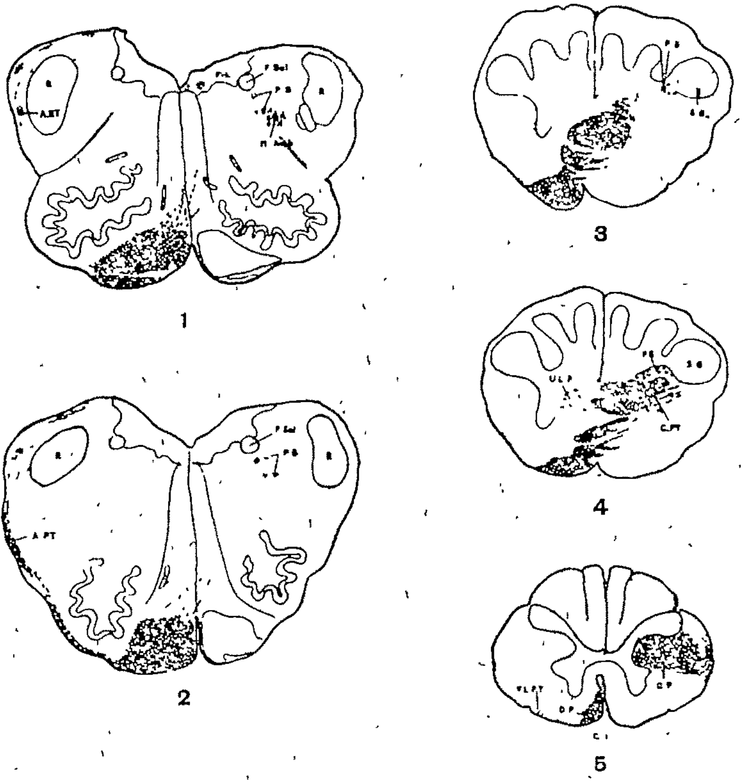


Fig. 4 shows the origin of Pick's bundle, which passes upwards and becomes scattered in the upper sections of the medulla (figs. 2 and 1). A few fibres only represent the ventro-lateral pyramidal tract (*V. L P. T.*, fig. 5) An aberrant tract (*A. P. T.*) arises from the pyramid in 2 and passes upward to reach the floor of the fourth ventricle (fig. 1). Many fibres leave the pyramid in figs. 1 and 2 to pass through the interolivary layer to the hypoglossal nuclei.

level of the decussation of the trochlear nerves, about one-fourth of the fillet fibres were degenerated. The arrangement of the fibres into bundles, their mesial direction and distribution to the motor fifth and seventh nuclei of both sides were all well marked and need no special description. No fibres could be traced to either hypoglossal nucleus.

Pick's bundle was very well marked in this case. The fibres first begin to appear (on the side opposite to the degeneration) in the lowest sections of the pons, and are situated just dorsal to the facial nucleus, between it and the substantia gelatinosa. Here only a few scattered fibres can be seen, but further downwards the bundles progressively increase in size and compactness, and lie between the nucleus ambiguus and the fasciculus solitarius, a little ventral to the latter (case 5, figs. 1 and 2). At the level of the decussation of the fillet, five or six well-marked bundles are seen close together just mesial to the substantia gelatinosa, and at the decussation of the pyramids the bundles—still not completely fused together—become lost in the crossed pyramid. For several sections, however, they can be traced as definite bundles and in transverse sections are cut obliquely just before they are lost, giving the appearance of partly turning inwards towards the direct pyramid of the opposite side at this point. In this case, more than in case 1, the appearance is that of a tract arising by the curling upwards of fibres just crossed over from the opposite side, and being distributed to the nucleus ambiguus and adjacent nuclei (? facial nucleus) of the same side. In no section could any degenerated fibres be seen crossing from the opposite fillet or pyramid to Pick's bundle, and as nearly every section was so examined, and the degeneration was very well marked, it seems certain that very few, if any, of the fibres could have had such an origin.

The ventro-lateral pyramidal tract was represented by a few scattered fibres only in the first cervical segment; none were seen below that point.

Another curious set of homolateral fibres was given off from the pyramid just above the middle of the olive (case 5, fig. 2). They passed upwards and outwards as a thick band of degenerated fibres round the olive with the external arcuate fibres to reach the region of the restiform body, around which they became scattered, the most prominent bands of fibres being situated near the nucleus cuneatus; two small bundles, however, were almost superficial, and lay close to the surface of the fourth ventricle, and could be traced nearly to the hypoglossal nucleus (case 5, fig. 1).

The crossed pyramidal tract showed no exceptional features, the degeneration at the edge of the cord outside the direct cerebellar tract being well marked in the cervical region.

The direct pyramidal tract was of average size, and could be traced to the lumbar enlargement.

The *lateral uncrossed* fibres came off from the decussating pyramid as a well-marked but rapidly diffusing tract (case 5, fig. 4).

THE VENTRO-LATERAL PYRAMIDAL TRACT.

It will be seen that I have been able to trace in four cases of hemiplegia in man a tract which descends with the pyramid from the cerebral cortex or basal ganglia, at least as far as the pons, and which in the spinal cord is situated in the ventro-lateral region near the periphery; and I venture to suggest that the term "ventro-lateral pyramidal tract" is the most convenient terminology, as showing both the immediate origin and the ultimate position of the tract. This tract consists of a number of small and large fibres which split off as a compact bundle from the pyramid, either in the lower part of the pons or in the medulla, and coursing obliquely outwards come to lie at the edge of the cord in the ventro-lateral region. Occasionally (case 3) they are not split off until after the decussation of the pyramids, and in this case the tract is formed from the direct pyramidal tract of the same side. No case is recorded in which the fibres are given off below the first cervical segment. In all cases the tract is an uncrossed one.

This tract is variable, not only in its size, but in its region of distribution. In most cases the fibres begin to become diffuse at the third cervical segment, and they cease to be a tract in the lower cervical region; but sometimes, as in cases 2 and 3, they can be traced down to the lumbar region. As a rule it is not more than about one quarter of the size of the direct pyramidal tract, but like the latter it is very variable; it may be completely absent, or as in case 2 it may be almost as large as the direct pyramid itself. For this reason it has been usual for those investigators who have recognised its origin to refer to this tract as an "aberrant" one, but as some fibres of it at least seem to exist in the cervical region in nearly every case of cortical and sub-cortical hemiplegia (as in four consecutive cases of hemiplegia reported in this article), and as in many, if not the majority, of such cases it constitutes a definite tract, I think this appellation is hardly justified, and it would be better to recognise it under the term "tract" as a definite branch of the pyramidal system in man.

The exact origin of this tract I have been unable to determine. From two of my cases it is evident that the fibres lie dorso-lateral to the main mass of the pyramid in the lower pontine region, and supposing, as the sections appear to show, that no serious rearrangement of fibres takes place higher up in the pons, it is probable that they are closely associated with the accessory fillet between the pyramidal and the temporo-pontine fibres in the crus cerebri. Beyond this region it is impossible to trace them: but it is quite evident that they are derived either from the Rolandic or closely adjacent cerebral cortex, or from the caudate or lenticular nuclei: they certainly do not arise in the optic thalamus. I am inclined to think that they have a cortical origin, but of this I have no actual proof.

Many cases have been reported in the literature in which it is evident that some of the fibres seen degenerated belong to this tract. *Meyer*,[1] in 1882, describes a case of extensive pontine hæmorrhage in which there was degeneration in the ventro-lateral region of the cervical cord. From his drawings it is probable that the vestibulo-spinal tract, the thalamo-spinal, ponto-spinal and some pyramidal fibres go to make up his degeneration, which was well-marked by the Weigert method. For fine degeneration in this region, however, the Weigert and carmine methods are of little value, because in the upper cervical segments the ventro-lateral region does not stain so deeply as the other columns; this appears to be partly owing to the fineness of many of its fibres and partly to the greater amount of interstitial tissue present here. This area which, like the tract under discussion, is triangular in shape, has been called the "Dreikantenbahn" by *Helweg*,[2] and, as is well figured in Bruce's "Atlas of the Spinal Cord," represents a normal structure. The recent work of *von Becterew*[3] shows that these fine fibres are derived from the inferior olive (the "fasciculus peri-olivaris"). It is therefore clear that the Marchi method alone is of value as indicating the presence of comparatively slight degeneration in this region, and it is of little use to speculate upon the value of the older observations in connection with this tract.

In several recent papers reference has been made to the fibres. *Risien Russell*,[4] in 1898, in a case of left hemiplegia due to sarcoma of the right cerebral hemisphere, figured a small tract at the decussation of the pyramids lying lateral to the pyramid itself: the fibres were distinct from the degenerate pyramid, "but did not appear to be derived from this source." Unfortunately sections through the pons are not figured; but the fibres occupied the ventro-lateral column of the cord, and there is little doubt that this was a ventro-lateral pyramidal tract.

Mott and Tredgold,[5] in 1900, described and figured, in a case of thrombosis of the middle cerebral artery, a tract in the ventro-lateral region of the uppermost cervical segments, and traced it as far as the lumbar enlargement. They say: "We are inclined to think . . . that its probable origin is the optic thalamus; that it passes through the middle fillet in the pons and the interolivary lemniscus in the medulla to reach the peripheral part of the antero-lateral column of the cord, in which it descends as far as the lumbar region." This suggestion was, the authors say, made after consideration of the results of Hoche's[6] work, to which reference will be made later. The pons and medulla do not appear to have been available for Marchi sections. I cannot think that the tract seen in the ventro-lateral region had this origin or course. In *Hoche's*[6] cases, as in one of mine (case 3), it is true that there was degeneration in the fillet in the interolivary region; but Hoche figures no degeneration in the ventro-lateral region of the cervical cord, nor could any of these fibres in case 3 be traced to this region; and in cases 1, 2 and 4 there was no interolivary degeneration and yet a well-marked ventro-lateral tract existed. The recent work, too, of *Collier and Buzzard*[7] shows that the thalamo-spinal tract in man, although corresponding fairly closely with this tract in the cervical cord, lies in the tegmental region of the pons dorso-lateral to the middle fillet, and that to reach this position it passes outside the olive and not through the interolivary fillet. Although, therefore, I cannot deny the possibility of the thalamic origin suggested by Mott and Tredgold for the tract in their case, the evidence is all in favour of its having had a pyramidal origin and of its having been independent of a possible interolivary degeneration.

Spiller,[8] in 1899, in a case of hæmorrhage into the lenticular nucleus and external capsule, also found this tract. In his case the fibres, separated from the pyramid in the lower pontine region, passed downwards amongst the fibres of Gowers' tract opposite the olive. They were not traced beyond the first cervical region as the rest of the cord was not available. He concluded that they arose either in the Rolandic area, the lenticular nucleus or optic thalamus, and tended to the view that they were not an "aberrant" but a constant tract.

Mme. Déjérine,[9] in 1900, described fibres in the ventro-lateral region of the cord in hemiplegia due to cortical lesions. These "fibres pyramidales homolatérales superficielles" arose from the pyramid at the middle of the medulla and passed outwards in front of the olive to reach the ventro-lateral position. The author does not state in how many cases these fibres were seen, but refers to them as "aberrant."

EMBRYOLOGY.—Many of the fibres of the ventro-lateral region of the cord become medullated very late—at about the same time as the pyramidal tracts (Barker,[10] von

Becterew[3]). I have confirmed this observation in the cord of a new-born child, but was unable to pick out the various sets of fibres which probably make up the ventro-lateral column. The late development of this region, however, suggests a similarity in function and origin between its fibres and those of the pyramidal tracts.

COMPARATIVE ANATOMY.—I am indebted to Mr. Victor Horsley for a photograph of the pons and medulla of a badger, in which a band of fibres is seen to be passing superficially from the mesial part of the medulla to the ventro-lateral region, just at the lower edge of the pons.

Schäfer[11] states that in the monkey a few fibres of the ventro-lateral column degenerate after lesions of the opposite cerebral hemisphere. ***Mellus***[12] does not mention any such fibres as degenerating after cerebral cortical lesions in the monkey.

I can find no other references to the existence of a ventro-lateral pyramidal tract in lower animals, despite the number of cases in which extirpations of the motor cortex have been done. It is well known that the direct pyramidal tract is usually small and often absent in monkeys, dogs and cats, and that even in man its variations are extreme, so that there is nothing surprising in these observations about the smaller and probably later-developed ventro-lateral pyramidal tract. The ventro-lateral columns of the cervical region in man are now known to contain the following sets of descending fibres:—

(1) Intersegmental fibres, short and long, originating in the spinal cord.

(2) The fasciculus peri-olivaris (von Becterew),[3] arising in the inferior olive.

(3) The vestibulo-spinal tract (Ferrier and Turner,[13] Risien Russell[4]) from Deiters' nucleus.

(4) The ponto-spinal tract from the pontine nuclei.

(5) The colliculo-spinal tracts (Boyce,[14] Collier and Buzzard[7]) from the corpora quadrigemina.

(6) The thalamo-spinal tract (von Monakow,[15] Boyce[16]) from the optic thalamus: and we may add

(7) The ventro-lateral pyramidal tract, probably from the cortex, possibly from the lenticular nucleus.

Of all these sets of fibres the last to be developed, both phylogenetically and ontogenetically, are the pyramidal fibres, and it is therefore not surprising that they occur only in the highest primates and should in them be variable.

THE ACCESSORY FILLET.

This name was given by *von Becterew* to a bundle of fibres which lying lateral to the pyramid in the crus cerebri leaves it in the mesencephalon, and coursing dorso-laterally through the outermost cells of the substantia nigra passes into the middle fillet in the upper pontine region. *Schlesinger*[17] called it the "lateral pontine bundle." *Hoche*[6] also described and figured it in two cases, and his excellent figures of the middle fillet at birth prove that fibres which degenerate in hemiplegia are of much later development than the rest of the fillet fibres. That the accessory fillet is distributed to the motor nuclei of the trigeminal and to the facial nuclei of both sides I can confirm, but I have been unsuccessful in tracing any fibres to the hypoglossal nuclei. No fibres could be traced to the third, fourth or sixth nerve-nuclei, and in this respect my results agree with those of von Becterew, Hoche, Schlesinger and Collier and Buzzard.

Most, if not all, of the degeneration seen in the interolivary layer in my cases (except case 3) was due to fibres of distribution from the pyramid to the nuclei of the hypoglossal and possibly to the adjacent nuclei: I could not trace any fibres from the accessory fillet as far as this region, and on this point disagree with the findings of Hoche. In case 3 the fibres seen in the interolivary region were largely derived from the posterior longitudinal bundles, but probably here also none were derived from the accessory fillet.

It is interesting to note that the accessory fillet receives accessions of fibres from the most dorsal layers of the pyramidal fibres in the pons, and that these layers, if traced upwards, are seen to lie in the crus between the accessory fillet and the main mass of the pyramid.

I have been so far unable to determine the exact origin of the accessory fillet fibres, as in all my cases not only the Rolandic area (or its corresponding fibres in the internal capsule) was degenerated, but also the lenticular nucleus was more or less involved; the probability, however, is that the fibres originate in the cortex of or near the motor area.

The *mesial accessory fillet* of von Becterew (Spitzka's bundle), was degenerated in three cases (1, 2 and 3). In each the fibres passed from the pyramid in the crus to the inner end of the corresponding middle fillet; the number of fibres so supplied to the fillet was small compared with those coming from the (lateral) accessory fillet.

PICK'S BUNDLE.

This small tract was first described by *Pick*[18] in 1889. He described it as a rounded, well-marked bundle ascending from the lateral column of the cord at the upper limit of the decussation of the pyramids to end in or near the posterior column nuclei, and possibly sending some fibres to the cerebellum. He only observed it in one case, and was of opinion that it was a rare abnormality.

Hoche,[6] in 1898, found it degenerated in one of two cases of hemiplegia; he was of opinion that the fibres were descending, and that the bundle was formed by the union of prematurely crossed pyramidal and accessory fillet fibres in the lower pontine and upper medullary regions, and that, as these fibres descended, they were collected together into one or more compact bundles and fused at the decussation of the pyramids with the crossed pyramidal tract, of which it was thus a tributary.

The appearance of the bundle in cases 1 and 5 lead me to differ from Hoche. Although nearly every section was examined in the medulla and lower pons, I could find no sign of any degenerated fibres crossing from the opposite fillet or pyramid to join the bundle; and further, the appearance of the bundle as it joins the crossed pyramid strongly supports the view that it is with the latter that the crossing takes place. Further, in all the cases that have been figured, and in my own sections, there is a progressive diminution in the number of fibres as we proceed upwards, the main bundle at the same time being split up into smaller and smaller bundles, until no fibres can be seen in the lower part of the pons. I think, therefore, that the tract known as Pick's bundle has a cortical or closely allied origin, that the fibres of which it is composed descend with the pyramid and cross with the main mass of the latter at its decussation, and then curve sharply upwards to ascend to its distribution amongst certain of the medullary nuclei, more especially the nucleus ambiguus.

That this bundle is not of such a rare occurrence as Pick suggested is quite evident, as I have found it in two out of five consecutive cases of hemiplegia, and Hoche also found it in two cases. It must be remembered, however, that Pick saw it only by the Weigert method in an undegenerated condition, and from his figures it is clear that it was larger than in any of the cases since recorded.

From the position it occupies with regard to the nuclei of the tenth and eleventh nerves, and especially to the nucleus ambiguus, it would appear that fibres are being supplied in this way from the pyramidal system to the motor nuclei of the vagus and spinal accessory nerves.

I have examined two series of sections through human fœtuses—one at the eighth month and the other at full term. In neither could any fibres corresponding to Pick's bundle be seen myelinated, although in two adult medullas stained by Pal's method it was quite easy to identify certain fibres as belonging to Pick's bundle. In all probability it myelinates late, but owing to the uncertainty of finding it in a normal Weigert specimen, it is very difficult to prove this. *Ransohoff*,[19] in 1899, came to the same conclusion.

I can find no reference to any such bundle in the lower animals.

OTHER TRACTS.

With regard to the *pyramidal tracts* I can add little to the excellent description given by Risien Russell in 1898. My sections confirm his view of the origin of the homolateral fibres in the crossed pyramidal region, and there was nothing to suggest that *Rothmann's*[20] suggestion was correct, viz., that the homolateral degeneration was due to compression of a bundle of the fibres of the opposite (undegenerated) pyramid by swollen degenerated fibres at the decussation. In one of the cases a definite bundle was clearly seen arising at the caudal end of the decussation from a position which no fibres of the opposite side could assume. In only one of the five cases I have examined was there any degeneration in the opposite direct pyramidal region.

Fibres were seen going from the pyramid in the pons to the motor nuclei of the fifth, to the seventh and twelfth nuclei of both sides, chiefly the opposite; none could be traced to the third, fourth and sixth nuclei. This observation agrees with that of Hoche.

It is clear from my cases that in man, even when the optic thalamus is not involved, the *crossed pyramid* in the cervical region extends further forwards than is usually stated (see microphotograph), and that some of its fibres lie diffused on the surface of the cord separating Gowers' tract from the direct cerebellar tract. It will be noted that in one case (case 5) an "aberrant" tract of curious distribution was observed, which, branching off from the pyramid at the level of the middle of the inferior olive, passed round the latter with the external arcuate fibres to reach the region of the restiform body, around which some fibres passed along the floor of the fourth ventricle. Possibly this was an extra (uncrossed) source of pyramidal supply to the nuclei of the hypoglossal and vagus. I have been unable to find any reference to a similar tract in the literature, but the fact of its recurving brainwards is of interest, as it shows that there is nothing impossible in the suggestion I have put forward as to the origin of Pick's bundle.

CONCLUSIONS.

(1) In man the pyramid frequently gives off a ventrolateral tract. This tract may arise in the pons, medulla, or first cervical segment of the cord; it lies in the region of Helweg's "Dreikantenbahn," and is best marked in the first

two cervical segments; occasionally it can be traced down to the lumbo-sacral region.

(2) I confirm, in the main, the researches of Hoche on the "accessory fillet" in man, *i.e.*, that it leaves the pyramid in the upper pontine region, descends in the middle fillet, and supplies certain of the cranial motor nuclei.

(3) "Pick's bundle" is probably an ascending tract which arises from the crossed pyramid at the decussation, and forms at least part of the pyramidal supply of the nucleus ambiguus; it is fairly frequently degenerated in cases of hemiplegia, and is not of such rare occurrence as Pick supposed.

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EXPLANATION OF ABBREVIATIONS IN FIGURES AND PHOTOGRAPHS.

A. F. = Accessory fillet.
A. P. T. = Aberrant pyramidal tract, described on p. 490.
B. C. = Brachium conjunctivum.
C. P. T. = Crossed pyramidal tract.
C. T. = Corpus trapezoides.
C. V. P. F. = Crossed ventral pyramidal fibres.
D. P. T. = Direct pyramidal tract.
Dent. = Dentate nucleus.

E. A. N. = External arcuate nucleus.
F. P. = Fronto-pontine tract.
F. Sol. = Fasciculus solitarius.
I. O. = Inferior olive.
M. A. F. = Mesial accessory fillet (Spitzka's bundle).
M. F. = Middle fillet.
N. Amb. = Nucleus ambiguus.
N. Mot. V. = Motor nucleus of trigeminus.
N. R. = Nucleus ruber.
P. = Pyramid.
P. B. = Pick's bundle.
P. L. B. = Posterior longitudinal bundle.
R. = Restiform body.
T. P. = Temporo-pontine tract.
U. L. P. = Uncrossed lateral pyramidal fibres.
V. L. P. T. = Ventro-lateral pyramidal tract.
X. = Tract described on p. 471.
V., *VI.*, *VII.* = Fifth, sixth and seventh nerves.

The figures were outlined with the aid of the Edinger apparatus, and details filled in from microscopic examination.

ON THE STUDY OF TRUE TUMOURS OF THE OPTIC NERVE.

BY PROFESSOR A. PICK, OF PRAGUE.

THE last few years have seen the appearance in rapid succession not only of numerous isolated contributions but also of large comprehensive works on true tumours of the optic nerves. In face of what thus appears to be the defined position of teaching with respect to this condition, fresh observations have to prove their right to publication; the opinion that this can be allowed to a subsequently recorded case by the fact that from various clinical and pathological points of view it may properly be regarded as unique, induces me to publish the following one as shortly as possible.

On March 13, 1891, Victoria R., the wife of a labourer, was transferred from Professor Sattler's eye clinic to that for psychiatry, as she appeared to be abnormal mentally. The history of the case from the eye clinic was as follows:—

The patient, admitted there on November 10, 1900, stated that about five weeks before she had sustained an injury to the left temporal region by striking violently against a broad wooden peg in a stable. Before the injury she had always been able to see well, and the evening before had even threaded a sewing needle by moon-light; immediately after the injury a fluid began to escape in drops from the left nostril and continued to so for about a week. Before this the patient had never been ill and had given birth to two children; since the last confinement two years ago the menses had ceased.

The general bodily examination which, considering the state of the eyes, was conducted with especial regard to the nervous system, and also by the writer, showed nothing abnormal; uterine examination showed *post-partum* suprainvolution of the uterus; there was also constipation.

The report of the examination of the eyes runs as follows: Externally, no signs of inflammation, eye movements follow promptly as ordered in all directions, the left eye shows slight permanent divergence, pupils both dilated and fixed, the media clear. Right papilla hyperæmic, swollen; the swelling measures about two to three dioptres and spreads some way on to the retina; the hyperæmia is an arterial one, showing a large number of fine vessels on the papilla; the veins also are distended and show a very tortuous course, there are no changes in the retina. Left papilla greatly swollen, the swelling being unequally distributed, the upper half and a three-cornered prism of the lower half being prominent. The hyperæmia on this side is also mainly arterial, a network of the finest vessels being spread over the papilla, the veins are also greatly distended and tortuous, the swelling is limited to the papilla, so that it stands out steeply from the surrounding retina which is normal, except for some changes mentioned below. These changes consist of small yellow dots which lie in the retina, are found to be under the vessels and are scattered over the whole field of the retina, but especially outwards and downwards from the papilla arranged in a line like a rosary.

Examination of functions.—The patient's statements are quite untrustworthy; so much can only be ascertained with certainty: that the vision of the left eye is better than the right, and colours are more quickly recognised; after repeated attempts the patient occasionally recognised small objects such as the hand of a watch; the light sense could not be tested because the patient stated she saw absolutely nothing in the dark.

Rhinoscopic and aural examination revealed nothing more than slight swelling of the mucous membrane of the left nasal cavity.

From the case book of the eye clinic the following extracts are given:—

November 20.—Papillæ on both sides greatly swollen, the left more than the right, on the left side the dotting (stippling) is more evident, the right papilla is already dirty gray. Blue and yellow recognised with certainty, but patient has red-green blindness.

December 8.—Diminution of swelling of right papilla, with undoubted *transition into atrophy;* upper half of left papilla still much swollen, but not the lower.

December 16. — Right, no essential alteration; left, the swelling less, the lower half of the papilla is changing to a dirty colour.

December 27.—Patient has wandered at night, says the Virgin Mary has appeared to her and has ordered her poultices to the abdomen.

January 14.—Left papilla pale; right, the upper half swollen, only a thick net-work of dilated vessels being visible there, the stipplings are visible scattered over the whole of the retina Red-green blindness, blue and yellow well recognised, field of vision only examined with the hand, but appears from very untrustworthy answers not to be contracted.

February 1.—On both sides marked increase of atrophy; left papilla pale with a tinge of dirty yellow, vessels already narrowed; right, only above is swelling to be seen, the vessels still of normal size.

February 18.—Restless at night; says God has appeared to her. Complains of paræsthesia in the left leg and nape of the neck, disappearing after a laxative.

March 1.—Pronounced atrophy of both optic nerves, the transition from papillitis still visible in the left.

On March 13 the patient was, as already mentioned, transferred to the clinic for psychiatry as she was more frequently hallucinated and restless. There she was in the first few days scarcely approachable, prayed almost constantly, would eat nothing, reacted violently against any touch; in calm intervals protested against her transfer, at night was still very restless.

On March 17 it was at last possible to make a thorough examination. She gave correct information considering her general condition and history; her recent conduct she explained chiefly by hallucinations. The fresh examination, especially of the nervous system, revealed nothing abnormal except the optic nerve atrophy. Subsequently the patient was often restless, occasionally violent to other patients because they made a "noise and bad smell"; at times quiet, asking to be discharged, and saying if it is God's will she will soon be well; she asked to see an advocate in order to arrange some business.

March 24.—Ophthalmoscopic examination.—Papillæ white, arteries narrow. Can see fingers at one metre; complains of headache. It is noteworthy that at repeated examinations *the pupils now reacted both directly and consensually*, the right more quickly.

March 26.—At 10.30 p.m., she suddenly cried out, breathed noisily, became cyanotic and had shaking movements of the arms, after which she slept on into the next day. The attack was not observed by a medical man.

March 27.—Complaint of headache, mark of biting on right edge of tongue, the left upper extremity perhaps not so steady as the right when held out, face equal, tendency to foot clonus, the left slightly more so, *pupils not acting to light.* Enema, ice-bag. In the afternoon about seventeen epileptic seizures of varying intensity, passing one into another in quick succession; after these, greatly increased knee-jerk and brisk foot clonus.

March 28.—In spite of repeated enemata no action of the bowels. Patient stuporous. Chloral hydrate given per rectum, in consequence of further recurrence of status epilepticus, in which the patient died on March 30, the temperature having risen steadily to 39° C.

The autopsy performed the next day in Professor Chiari's anatomico-pathological institute gave the following results:—

Œdema of the brain, gray degeneration of the optic nerves, bilateral lobular pneumonia, bronchitis, obsolete tubercle at the pulmonary apices and in the peri-bronchial glands. The following passage from the report is noteworthy. *The optic nerves markedly gray in colour although not diminished in size.* If this was remarkable the microscopic examination was equally so. In the first place, examination of Weigert-Pal preparations showed that both optic nerves, the chiasma, and also the stump of the optic tract did not exhibit typical staining, so that an inexperienced observer would have made a diagnosis of atrophy of the optic nerves. A closer examination, however, showed that even with this stain and without regard to the incongruity already observed macroscopically between evident atrophy and absence of any wasting, there were found in the transverse section, beside numerous round cells, peculiarly shaped bodies of varying size furnished with long processes, and that moreover, the sheath of the optic nerve was considerably thickened. In spite of some difficulty which arose from the fact that the whole specimen had already been hardened for a long time in Müller's fluid it was attempted to make a teased specimen, which sufficed to show that in addition to the completely atrophied nerve fibres and a moderate thickening of the vessels there were on the one hand masses of round cells and on the other the typical or angular cells of myxoma with often extraordinarily long spirally twisted processes, thus marking the tumour as a myxosarcoma.

The special points of this case are so evident that they only need a short summary.

Before all, stands the question of diagnosis; tumour of the optic nerves was not diagnosed. A latent affection of brain in connection with the suprainvolution of the uterus was much more considered, indeed, the absence of a diagnosis still seems justified, Braunschweig, who recently collected cases of genuine tumour of the optic nerve (*Graefe's Archiv.* Bd. 39, Abth. 4, p. 24), having found that exophthalmos is never absent in such cases; our present case is, however, appropriate to this point, and shows the importance of the little word "never," already discarded in medicine, being limited in its applicability.

The present case should also cause *the early development of visual defect* to be regarded as of diagnostic importance, inasmuch as the *contrast to the condition in choked disc the result of cerebral disease* compels us to lay important stress on the direct disturbance of the optic nerves caused by the infiltrating tumour. Our case emphasises and confirms the statement of Braunschweig (*loc. cit.*, p. 36): "If characteristic ophthalmoscopic appearances for optic nerve tumours are not found, still the regular early occurrence of inflammation and swelling of the optic disc is conspicuous, and where palpation does not give evidence of a tumour and exophthalmos has not developed, a tumour of the optic nerves should occasionally be borne in mind, and at least a guarded prognosis should be given."

Noteworthy also is the *etiological impetus.* Considering the absence of any characteristic symptoms of tumour of the optic nerve, one was inclined in the present case to regard the trauma as the result of an already present but unobserved defect of vision; in this direction also the case may be instructive in that trauma is so often given as the cause of tumour of the optic nerve. Important also is the rapid course of our case in contrast to those previously described. Whether the slight strabismus observed, as in other similar cases, was due to the tumour must remain undecided in the absence of a reliable history. Further, the very remarkable facts with reference to the variable condition of the reflex pupillary reactions must be referred to. From the importance of this symptom one is reminded that similar symptoms in cerebral syphilis depend on the state of syphilitic granulations due to changes in nutrition resulting from vascular fullness; and in so far as in that condition the groundwork of the tumour growing in the optic nerves consists of a soft tissue rich in cells, we are perhaps, not wrong in giving a similar explanation here. To further careful observations must be left the decision as to how far this symptom may be of diagnostic value. Finally, stress must be laid on the fact that perhaps initial palpation of the optic nerves is of diagnostic value as inspection of the same would never have led to a diagnosis. We are therefore brought to the pathological anatomy of our case, which certainly is of no small interest. Firstly the position and extent of the tumour appears to me important, for as one cannot suppose that both optic nerves were separately and simultaneously diseased we are driven to the conclusion that apparently the chiasma was the original seat of the growth and that thence the spread took place on the one hand into both nerves and on the other into the tracts; secondly, the absence of any tumour bulging out the optic sheath from within appears to me to be important considering its longitudinal extent; further the absence of any other abnormality in the shape of the optic nerves which so often in similar cases are bent like a post-horn. One can only suppose that the thickened sheath opposed a considerable resistance to the transverse growth of the mass of the tumour, and therefore led to a marked development in the long axis and so to speak to a purely infiltrating growth. Perhaps the unusually long extent of the tumour through the optic nerves and chiasma is thus to be explained.

Lastly, a few words as to the symptoms indicating the participation of the brain, which I think may be partly explained by what was found. First as to the psychosis which apparently was based essentially on hallucinations of sight. These corresponded undoubtedly with what one designates pure hallucinations of sight as they are seen in disease of the retina or optic nerves, this is confirmed by the subsequent occurrence also of hallucinations of hearing and smell. If the disease of the optic nerves was related to the first of which indeed it appears to have been the basis, it is perhaps not impossible that pressure of the tumour on the nerve trunks was partly responsible for both the latter. As to the meaning of the epileptic seizures, it must be remembered that in a perfectly unique case of Alt's (quoted by Braunschweig, *loc. cit.*, p. 43), epileptic attacks were also observed as the case developed; Alt explains their occurrence by pressure on the parts of the central nervous system lying adjacent to the chiasma, but it appears to me that the facts point rather to the influence of a disturbance of the state of the circulation in the dural sheath of the optic nerves compressed from within outwards.

A CASE OF CEREBRAL AND CEREBELLAR TUMOURS WITH WELL-DEFINED TRACT DEGENERATIONS.

BY F. THIELE, M.D.

Pathologist to University College Hospital, &c.

A. B., aged 6 years, was admitted into University College Hospital on April 19, 1900, under Mr. Horsley.

(*a*) *History on admission.*—The patient was sent by Dr. Wohrnitz with the following history:—

On March 17, five weeks previous, the boy fell out of a mail-cart on to the raised edge of a stone from which some railings sprung, *i.e.*, a sort of kerb stone, striking the occipital region. The child cried and a swelling formed on the back of the head as a result of the contusion. Nothing was noticed till the following Sunday, March 19, when the patient became sick and "rigid as if in a fit." He was thereupon sick on and off until admission.

On Easter Monday, April 16, the patient seemed to have a fit, a right-sided paralytic stroke affecting the entire right side, arm and leg. This passed off.

A fortnight ago, *i.e.*, about April 5, the child commenced to lose sight in both eyes and for the last week he was completely blind in both eyes: prior to the accident he had been able to see quite normally. There was no difficulty in swallowing, &c.

When he had been sitting quietly for a few minutes the mother noticed his eyes to be in a condition of marked convergent strabismus.

Present state.—The patient is a well-nourished child in rather a stuporous condition. When he sits or stands the head is held in an oblique direction so that the face is directed towards the left front, the right ear being slightly approximated to the right shoulder.

He stands steadily, but walks with the legs separated and with a cerebellar gait.

Head.—Both a subjective sensation of pain and local tenderness exist in the occipital region. The head appears rather large.

Cranial nerves.—(I.) Not tested. (II.) Double optic neuritis. Rather more marked (observation by Mr. Percy Flemming) on the right side than the left. There were no hæmorrhages, but on both sides numerous brilliant white spots. (III., IV., and VI.) (1) Extra ocular movements. Though there was no true nystagmus both eyes tended to swing towards the left. (2) Intraocular movements. At rest the pupils were equal and dilated. They varied from time to time, but the contraction was feebly marked. Remaining cranial nerves normal.

All movements of the limbs and trunk could be performed though feebly.

Reflexes.—*Superficial.*—There was strong plantar flexion of the great toe. *Deep.*—Knee-jerks: right very feeble, left present. Examined on another occasion by Dr. Risien Russell they were found to be about equally active. Triceps-jerks present and equal.

It was decided to open the dura mater over the cerebellum, but when this was done the brain bulged greatly and severe hæmorrhage occurred from a vein at its entrance into the torcula Herophili.

This was ultimately arrested and the wound closed, but the child died subsequently from shock.

At the autopsy the following tubercular lesions were found:—

Seat of tumours.—(1) In the upper third of the left parietal lobe there was a tumour extending from the plane of the end of the lateral ventricle in front to the plane of commencement of the three zones of the globus pallidus posteriorly.

(2) There was a much smaller tumour extending from the splenium to the hinder end of the parietal tumour.

(3) In the superior temporal convolution at its hinder end and occupying its breadth was a small tumour about 1 cm. in diameter.

(4) In the ventral half of the left lateral lobe of the cerebellum was a large tumour.

A large series of microscopic sections was made through the cerebral hemispheres and neural axis, stained by the

Marchi method. The results obtained were so definite as to afford a contribution towards the correct topographical delimitation of certain tracts.

Sub-cortical fibre systems.—The corona radiata of the parietal lobe of the left hemisphere showed wide-spread degeneration of fine collateral fibres and immediately under the cortex degenerated collaterals were given off, which ran horizontally and others which ran from one gyrus to the other, running around and beneath the sulcus. Deeper down in the corona radiata strong collaterals were seen coming off from the pyramidal axones which could be traced to the corpus callosum.

Corpus callosum.—In the frontal region the corpus callosum showed no signs of degeneration. At the level of the middle of the third ventricle the corpus callosum showed degeneration in two groups—a dorsal layer in which the fibres were mixed, *i.e.*, both coarse and fine, and a ventral layer in which the fibres were all coarse. The fine degeneration barely extended to the vertical plane of the opposite cingulum—the coarse fibres however passed on through the corona radiata and were distributed to the cortex of the opposite parietal lobe. No fibres were seen to turn downwards and the general horizontal level maintained by the callosal fibres was that of the fasciculus subcallosus.

The fasciculus subcallosus was not degenerated in the right hemisphere but was extensively so in the left and fibres (? collaterals only) appear to pass from it into the optic thalmus for a short distance.

Temporo-thalamic fibres.—Entering the left thalamus on its outer side were numerous bundles of temporo-thalamic fibres which crossed the descending pyramidal fibres and which were of a much finer calibre. These fibres penetrated the optic thalamus for two-thirds of its width and there broke up first internal to the lamina medullaris interna. They traversed the corona radiata dorsally to the islands of gray matter of the posterior part of the lenticular nucleus running downwards and occupying most of the internal capsule till they approached the coarse pyramidal fibres from which they were sharply distinguished.

Habenular ganglion.—There was a fine degeneration in this ganglion and in the tænia thalami. Though there was no degeneration in the crura of the fornix there was a similar fine degeneration in the capsule of the corpora albicantia. These areas of ventricular degeneration are to be attributed to the intraventricular pressure common in such cases and marked in the present instance.

Pyramidal tracts.—Numerous degenerated fibres of a coarse calibre descended on the left side from the parietal cortex, *i.e.*, the lower limb area. These fibres passing down in the corona radiata and opposite the middle of the optic thalamus occupied a more ventral position in the corona radiata. The fibres, from the marginal aspect, partly crossed in the corona radiata those coming from the dorsum of the hemisphere. At the level of the posterior commissure the degenerated fibres occupied the following positions in the crusta; thus of the whole crusta in this region there remained free from degeneration the outer one-seventh and the inner three-sevenths, and in the more mesial portion of the crusta the degenerated fibres are almost wholly confined to the ventral border.

A few scattered degenerated fibres could be seen in the substantia nigra.

In the pons it was seen that certain pyramidal bundles were wholly free from degeneration, viz., the most ventral and external bundle and the most mesial bundles. The mesially placed bundles in the pons rapidly decrease in size —the diminution being probably due to giving off of fronto-pontine (Flechsig), or perhaps more correctly thalamo-pontine, fibres—such pontine fibres being quite free from degeneration.

The degeneration is then traceable through the medulla to the decussation where the majority of fibres cross to the crossed pyramidal area of the right side and a few are continued down in the anterior direct pyramidal area of right side.

Fillet.—In these sections there is also to be seen some degeneration in the fillet on both sides. The degeneration is first seen in the middle region of the pons and can be traced as far as the corpora quadrigemina inferior in which the fibres become lost. In the lowest part the degeneration is most marked in the outer part of the median fillet and when the fillet is completely differentiated the degeneration is seen to be most marked in the lateral fillet.

"*Accessory fillet.*"—This is well marked in this case as a few coarse scattered fibres arising from the outer part of the pyramidal bundles, crossing the band of gray matter which separates these from the fillet and entering the latter in the outer part of the mesial fillet, *i.e.*, in the lateral angle that the mesial and lateral fillet make with each other.

Optic tracts.—Both of the optic tracts show marked degeneration, and as they pass backwards no fibres are given off, to the crusta or pass from that round to the corpus subthalamicum (Luysii). The majority of tract fibres pass into the corpus geniculatum externum, but the most posterior and inner enter the optic thalamus and pass inwards in two sets, ventral and dorsal.

Posterior commissure.—There is heavy degeneration in both divisions of the posterior commissure.

Posterior longitudinal bundle.—This was degenerated on both sides. The degeneration could be traced upwards as far as the plane of the middle of the red nucleus. The fibres of the posterior bundle show distinct decussation near the origin of the third nerve.

Spinal cord.—In the spinal cord there is no degeneration at all in the region of the so-called cerebellar tract. The region of the antero-lateral descending cerebellar tract is quite free from degeneration. The only degeneration seen in the cord in addition to the pyramidal is a marked coarse degeneration in both columns of Burdach. This degeneration can be traced upwards as far as the sensory nuclei of the medulla, beyond which it cannot be traced. The nuclei themselves appear to be quite normal. This degeneration may possibly be due to meningitic changes, but on this point of posterior column degeneration occurring in association with encephalic tumour, reference should be made to the valuable paper by Collier and Batten, BRAIN, 1900, p. 39. In the medulla degeneration is more marked on the left than right side. No degenerated fibres can be seen to run from the olivary nuclei to the opposite side to enter the restiform body, and similarly none can be traced from the sensory nuclei of medulla crossing to opposite side, but the medulla itself is grossly distorted by the growth in the left cerebellar lobe, there are some degenerated fibres among the right superficial arcuate fibres.

There was no degeneration in Deiters's nucleus on either side nor was there any degeneration in relation with it, Rollers' bundle presented no degeneration.

Cerebellum.—There was partial degeneration of the superficial pontine fibres leaving the cerebellum. In the cerebellum there was heavy degeneration in both flocculi, the uvula and inferior vermis as well as the fleece fibres around the dentate nuclei of the cerebellum. The degeneration was principally on the left side. There was degeneration about the roof nuclei and on the left side the degenerated fibres formed a distinct bundle running beneath the ependyma. The ventral commissure was also degenerated. The degeneration of fibres around the right dentate nucleus occurred in the form of a funnel shaped area originating (1) outside the nucleus; (2) from hilum. Both superior cerebellar peduncles were degenerated but especially on the left side, The degenerated fibres could be traced to the middle of the red nucleus. In the left red nucleus the degeneration was speedily lost, and higher up no degeneration could be made out in Forel's area.

On the right side the degenerated fibres soon became lost in the red nucleus, but a fine degeneration could be traced passing the inner aspect of the red nucleus into Forel's area. The degeneration in this case passed to the inner side of Vic d'Azyr's bundle and was lost in the thalamus. The red nucleus on either side is quite normal in size and extent.

It appears justifiable to draw the following conclusions from this case :—

(1) *With destructive lesions of the cortex of the Rolandic area.*—(*a*) The association fibres of the homolateral hemisphere undergo extensive degeneration. (*b*) The fibres of the corpus callosum connecting the two areas are of medium

size and do not turn down into the capsule of the opposite hemisphere. (*c*) The pyramidal fibres give off collaterals in their course through the corona radiata. (*d*) The fibres from the upper third of the Rolandic area run through about the junction of the middle and posterior thirds of the hinder limb of the internal capsule and for the most part in the outer side of the crusta of the peduncle, and maintain their relative position in passing through the pons.

(2) No fibres are given off by the optic tract to the infundibular region or to the corpus Luysii, in fact no fibres leave the tract before it reaches the level of the lower border of external geniculate body. This is contrary to the views of Bechterew, Stilling and Kolliker.

(3) Although there was widespread destruction in left lobe of the cerebellum there was no descending cerebellar degeneration in the spinal cord. This is contrary to the opinion of several authorities, *e.g.*, Marchi and Biedl; but inasmuch as Deiters's nucleus was intact the present observation supports the views of Risien Russell, and Ferrier and Turner.

(4) The accessory fillet is a descending tract contrary to the opinion of Bechterew, Schlesinger and in support of the view of Redlich and Hoche. The "accessory fillet" may be simply fibres of the pyramidal tract leaving to gain cranial nerve nuclei.

Review.

Anleitung beim Studium des Baues der Nervösen Centralorgane im gesunden und Kranken Zustande. Von Dr. HEINRICH OBERSTEINER, K.K.O.O., Professor, Vorstand der neurologischen Institutes an der Universität zu Wien. 4th edition, enlarged and revised with 250 illustrations. (Franz Deutuka, Leipzig und Wien, 1901.)

THIS standard work, already familar to English readers in the excellent translation of an earlier edition by Dr. Hill, has now attained its fourth German edition. So much new material has, however, been added, and so many fresh illustrations inserted, that the work, now consisting of 663 pages, is thoroughly up to date and stands in the fore-front of modern neurological textbooks.

The book consists of seven divisions. In the first of these, the methods of examination of the central nervous system, both macroscopic and microscopic, are concisely described. It is recommended that alcohol as a primary hardening agent be avoided, preference being given to solutions of formol or of bichromate of potash. The subsequent embedding in celloidin (or, better, in photoxylin, a more soluble medium) and, when very thin sections are desired, in paraffin, is clearly detailed. Weigert's method of serial sections in celloidin is explained. The technique of all the more important stains is discussed, a useful series of laboratory formulæ is given, and a short account of *intra vitam* staining by means of methylene blue concludes the section.

The second division describes the gross or naked-eye anatomy of the spinal cord and brain. The numerous "skeleton" maps of various regions which illustrate the text will be particularly useful to the student for reference. The physiological functions of the various parts of the cortex are then discussed, particular attention being drawn to the fact that the various cortical centres are not sharply marked off one from another, but merely represent the positions of maximal physiological association with some particular function, so that the different areas overlap to a greater or less extent. The various "motor centres" are enumerated. The centre for synergic ocular movements is located in the angular gyrus. The striped muscles of the bladder and of the rectum are probably represented somewhere in the cerebral cortex, but no special "centres" can yet be assigned to them. Obersteiner does not discuss the "kinæsthetic" theory of the so-called "motor areas."

The third division of the book is devoted to the histology of the central nervous system. The neurone theory is perhaps somewhat too briefly discussed, considering that its position is at present so much the subject of controversy. Obersteiner is frankly utilitarian on the point and avows that "even if later investigations demonstrate the neurone theory to be untenable," yet the theory makes the intimate structure of the nervous system much simpler and easier of comprehension. A lucid description, illustrated by excellent figures, is given of the various types of nerve-cells and nerve-fibres, together with an account of the ependyma, the neuroglia and the connective tissue and blood-vessels of the brain and spinal cord.

The fourth section is devoted to the minute structure of the spinal cord. The characteristic appearances of sections at various levels are described, and a tabular list is given of the functions of each spinal segment, compiled from the tables of Starr and Edinger respectively. There are, unfortunately, no figures to illustrate the segmental cutaneous distribution, and the description is hardly full enough to compensate for this deficiency. The various tracts in the cord are then described. An account of the spinal blood-vessels follows, and this section of the book concludes with a useful *résumé* of the chief pathological changes met with in the cord.

The fifth section gives a general topographical account of the brain, the descriptions being amplified by a series of excellent "skeleton" maps.

In the sixth division of the book, the chief tracts in the brain and cord are discussed in greater detail. First the pyramidal tracts, the posterior columns and their connections, and the cerebellar tracts, dorsal and ventral, are described; then follows a clear and succinct account of the various mesencephalic tracts, including Monakow's bundle, the ventral longitudinal bundle, the posterior longitudinal bundle, and the central tegmental tract.

This difficult subject is handled in a masterly fashion. No fewer than ninety pages are devoted to a minute study of the cranial nerves and nuclei. The cerebellum with its central and cortical gray matter is then described, and the section is completed by an account of the basal ganglia, the various association systems of the brain, the variations in structure of the different regions of the cortex, and finally the structure of the pineal and pituitary bodies.

The seventh and last section deals with the cerebral and spinal meninges and with the arrangement of the larger cerebral blood-vessels.

To each sub-section of the book there is attached a copious bibliography.

Throughout the work the reader cannot fail to be struck by the admirable clearness of the diction with which Professor Obersteiner discusses his subject and by the broad-minded manner in which controversial points are approached and opposing views stated. As a practical anatomical text-book of great interest and in close sympathy with clinical and pathological work Professor Obersteiner's work is cordially to be recommended.

PURVES STEWART.

THE Index of BRAIN for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, is now in the press, and it is expected that it will be published by the end of this year.

The price to members of the Neurological Society will be 6s. 6d., post free, obtained from Messrs. MACMILLAN & Co., Ltd., St. Martin's Street, London, W.C. A form of order is enclosed.

To others than members of the Neurological Society the price will 8s. 6d. net, and the volume can be obtained through any bookseller.

As the cost of the Index has been considerable it is hoped that every member of the Society will order at least one copy.

EDITOR.

BRAIN.

PART IV., 1901.

Original Articles and Clinical Cases.

A STUDY OF THE DEGENERATIONS OBSERVED IN THE CENTRAL NERVOUS SYSTEM IN A CASE OF FRACTURE DISLOCATION OF THE SPINE.

BY F. H. THIELE, M.D., B.SC., M.R.C.P.

AND

VICTOR HORSLEY, F.R.S., F.R.C.S.

With Photomicrographs by E. J. Worrall, M.D.

From the Laboratory of Chemical Pathology, University College, London.

FOR the opportunity of recording the following case we are greatly indebted to the kindness of Dr. Cecil Lyster, under whose care the patient was treated at the Bolingbroke Hospital. As it was clear that the accident must have caused a complete destruction of the spinal cord at and below the level of the third lumbar segment, and as the patient survived for a period just within six weeks, it seemed certain that by application of the Marchi method it would be possible to trace completely, besides the posterior column fibres, the following tracts:—

(1) The direct cerebellar tract of Flechsig or the *fasciculus spino-cerebellaris dorso-lateralis.* (Barker.)

(2) Gowers's tract or the *fasciculus spino-cerebellaris ventralis.*

(3) The *fasciculus spino-quadrigeminalis.*[1] } Mott. Patrick.
(4) The *fasciculus spino-thalamicus.* } Tooth.

The following is a brief clinical abstract of the principal clinical facts of the case:—

Case of W. L., aged 21, *Bolingbroke Hospital.*

The patient was brought to hospital having been knocked down by an engine and crushed against the edge of the platform. On admission, besides (1) a severe scalp wound in occipital region, and (2) dislocation of the right astragalo-scaphoid articulation with a contused wound of left calf, there was (3) fracture dislocation of the spine in the lower dorsal region. There resulted immediate and complete paralysis and anæsthesia of both legs; all reflexes were absent, and the anæsthesia extended up to the umbilicus. Retention of fæces and urine existed from admission. Cystitis supervened, and though the wounds in the leg were actively treated they showed no tendency to heal. The wasting of both legs and thighs became extreme. No reflexes were ever obtained. The patient died forty days after the accident.

The brain and spinal cord were removed by Mr. H. J. Curtis, F.R.C.S., under difficult circumstances, and for whose assistance in this particular we are indebted. The central nervous system was fixed in formalin 5 per cent. solution and Muller's fluid in equal parts, and subsequently in Müller's fluid alone. For the osmic stain Busch's fluid was employed (solution of osmic acid in iodate of soda solution).

In the crushed area at the level of the fourth lumbar segment the vessels were found to be all thrombosed, the smallest capillaries being engorged with blood. There was also a large extravasation of blood outside the cord, and the crushed substance of the spinal cord had been squeezed upwards (*i.e*, headwards) beneath the arachnoid for two segments distance, so as to simulate the heterotopic appearance of a double cord.

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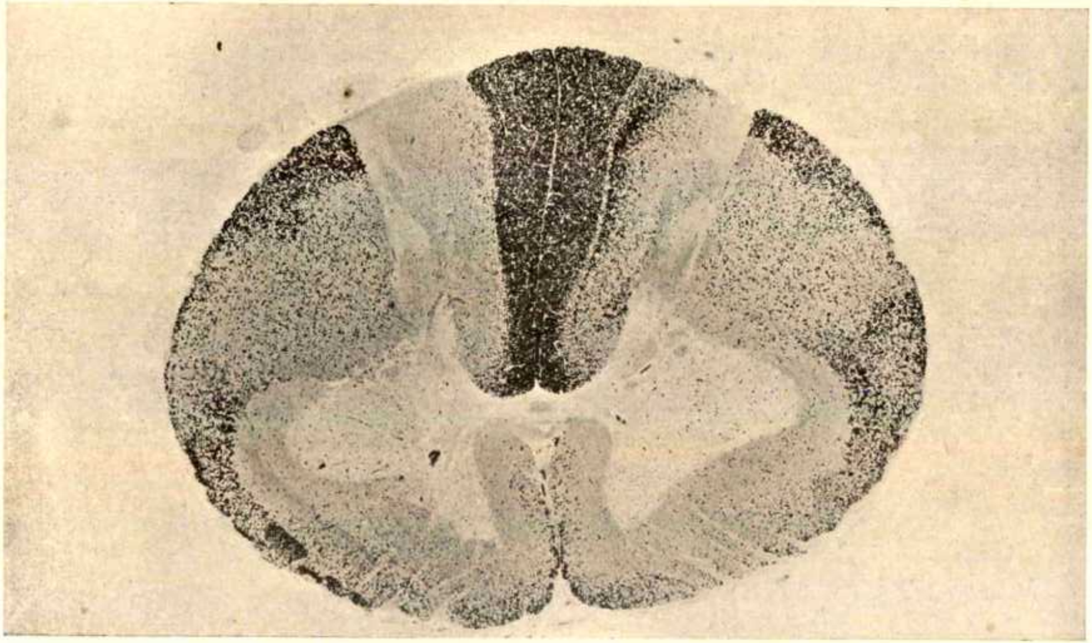
[1] We would support the use of the expression fasciculus spino-tectalis for this tract as being shorter and more in accord with general morphological terminology.



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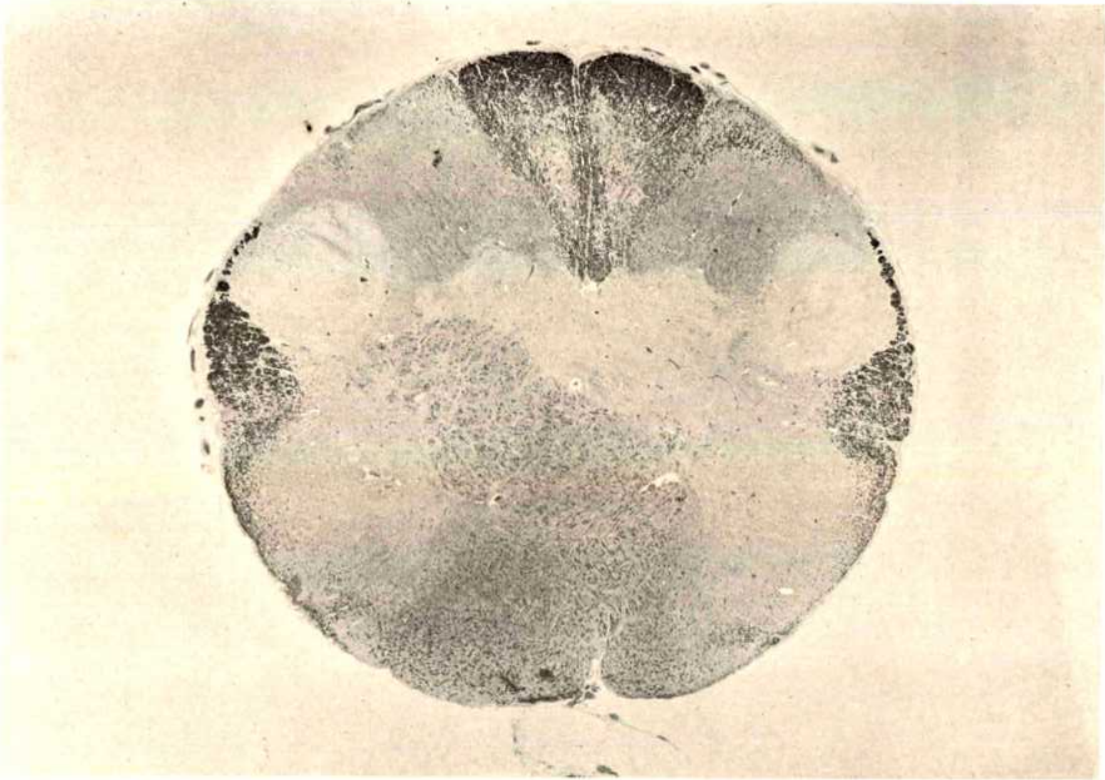

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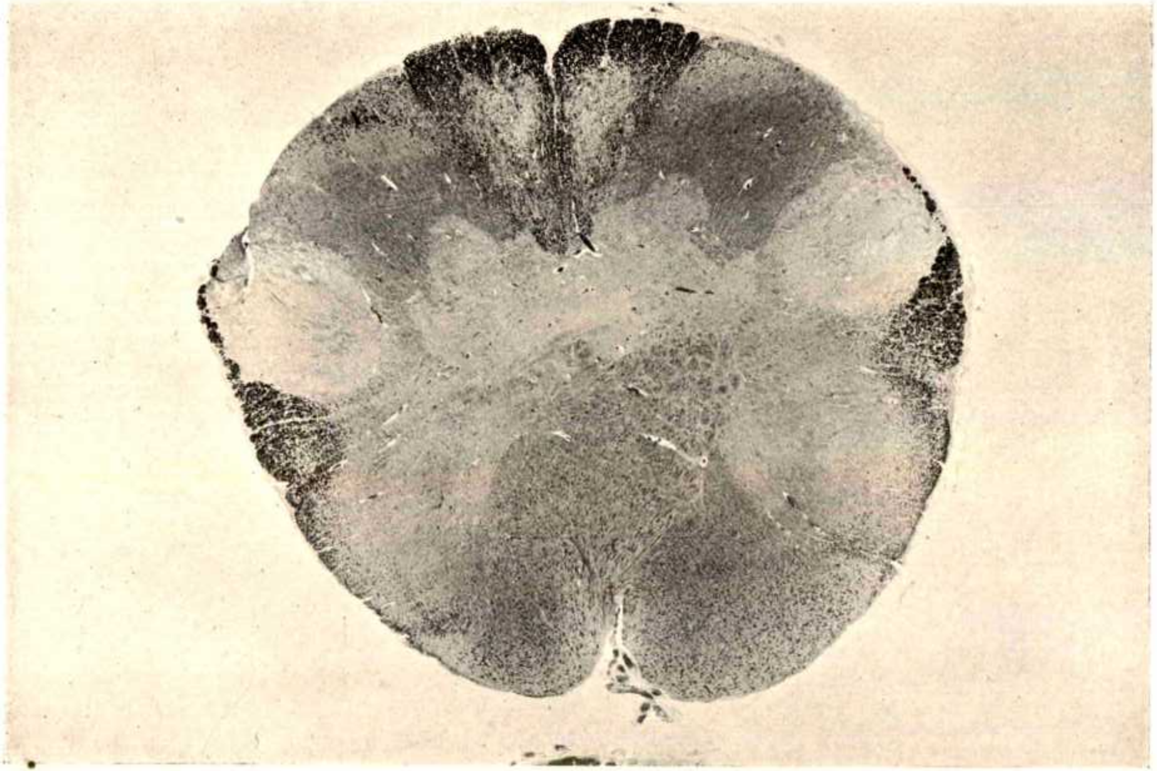

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(1) *The Direct Cerebellar Tract of Flechsig or the Fasciculus Spino-cerebellaris Dorso-lateralis.* (*Barker*.)

This tract is seen in the lowest sections as a bundle of coarse fibres symmetrically degenerated on either side of the cord. It has its customary situation, and extends forward to the ligamentum denticulatum, at which point it is continuous with the antero-lateral tract. At the level of the decussation of the pyramids the degeneration reaches dorsally to the inner border of the subst. gelatinosa on the one side but not on the other; it maintains this relation at the middle of the pyramidal decussation. When the restiform body is fully formed the direct cerebellar fibres occupy its anterior portion. In sections through the lower part of the bulb there are some coarse fibres to be seen lying ventrally to the spinal fifth root; these subsequently pass inwards and dorsalwards, and on examination prove to be only fibres of the direct cerebellar tract taking this somewhat unusual course. The coarse direct cerebellar tract fibres run straight upwards and backwards into the white matter of the cerebellum accompanied on each side by a broad band of fibres which are not degenerated, and which form the olivary division of the restiform body. These direct cerebellar fibres arrive opposite the lateral edge of the lowest folium of the vermis and there branch and subdivide. A few pass under the border of the vermis, but the large majority pass dorsally and spread out into the white matter, and ultimately pass across in the anterior commissure. As the fibres pass backwards they are so arranged that the olivary constituents of the restiform body run caudal to the spinal fibres. As they enter the cerebellum some degenerated fibres can be traced directly to the peduncle of the flocculus.

In the posterior commissure of the cerebellum only half the fibres therein are degenerated, the other (non-degenerated) half are therefore probably olivary in origin.

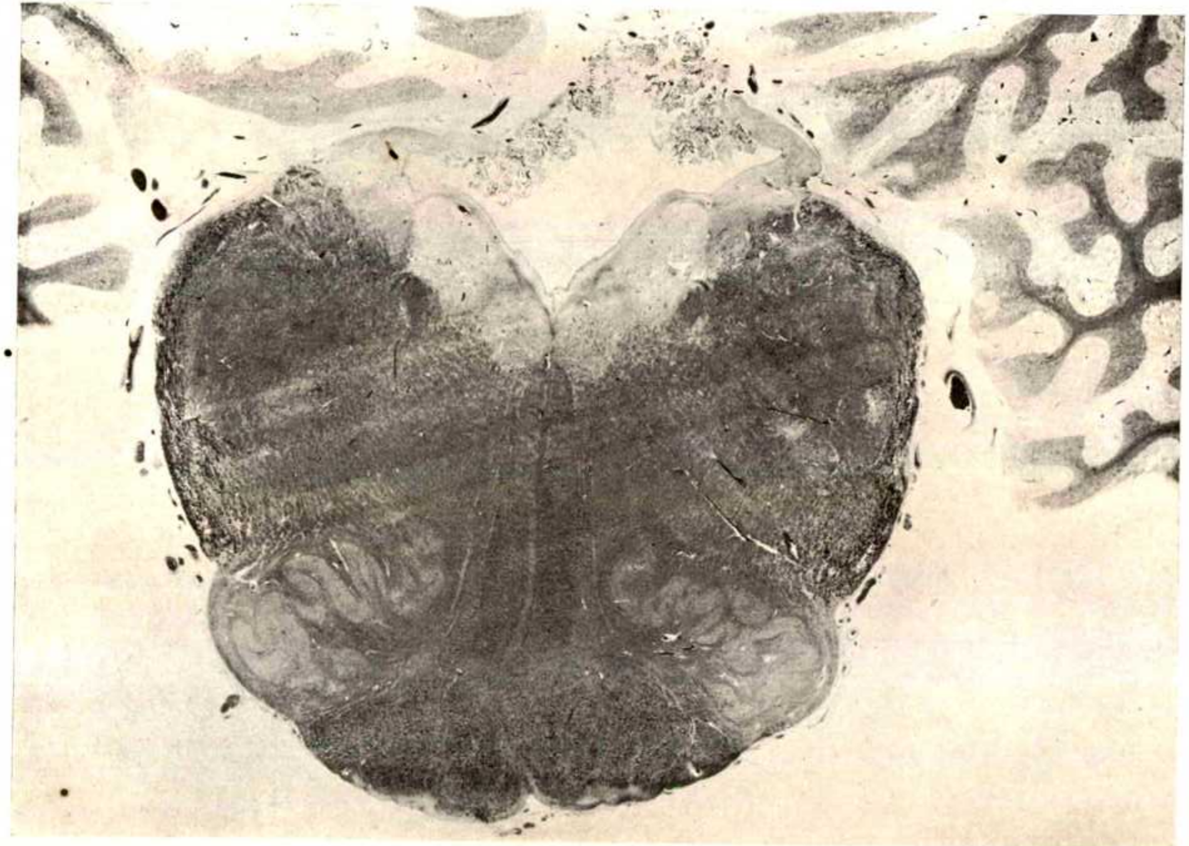
The direct spino-cerebellar fibres terminate mostly in the superior vermis, and are distributed in diminishing degree in the rest of the cortex cerebelli of each side, the fibres diminishing in number and calibre, so that the fibres in the posterior superior lobule are extremely fine, whereas those in the superior vermis are both fine and coarse.

The distribution of degenerated fibres to the cerebellar cortex is most marked in the ventral division of the lateral lobes.

So far the arrangement of the fibres observed corresponds with that usually described. We wish now to draw attention to the special relations of the direct spino-cerebellar tract to the structures in the lateral region of the bulb and termination of collaterals derived from the tract. These we will describe according to their morphological appearance, which is that of plexuses.

Fine collateral plexuses in and round the spinal root of trigeminus.—(*a*) *Ventral collateral plexus.* Just above the uppermost level of the decussation of the pyramids strong collaterals springing from the fibres of the direct cerebellar tract as they turn up towards the restiform body pass inwards just ventrally to the spinal trigeminus root and between that and the antero-lateral nucleus. This may be termed the ventral bulbar collateral plexus of the direct cerebellar tract. The fibres ultimately composing it are very fine, and appear to terminate in relation with a nucleus of small nerve corpuscles lying in a plane just internal to the ventral border of the spinal trigeminus root. The uppermost limit of this ventral plexus is the upper border of the auditory striæ. We have found a definite interruption in this plexus for some distance (from 1 to 2 mm.), and that it consequently reappears soon before it finally ends. The nucleus referred to reaches in the formatio reticularis from 1 to 2 mm. higher than the end of the plexus, and is, perhaps, the posterior subdivision of the nucleus lateralis.

(*b*) *Dorsal collateral plexus.*—Another plexus of fine collateral (?) fibres is seen to come from the direct cerebellar fibres, passing inwards and partly through the spinal fifth root. These are collected in a fine plexus just external to the gray nucleus enveloping the fasciculus solitarius, and is, of necessity, especially well-defined on the outer side of the gray matter surrounding the fasciculus solitarius. It



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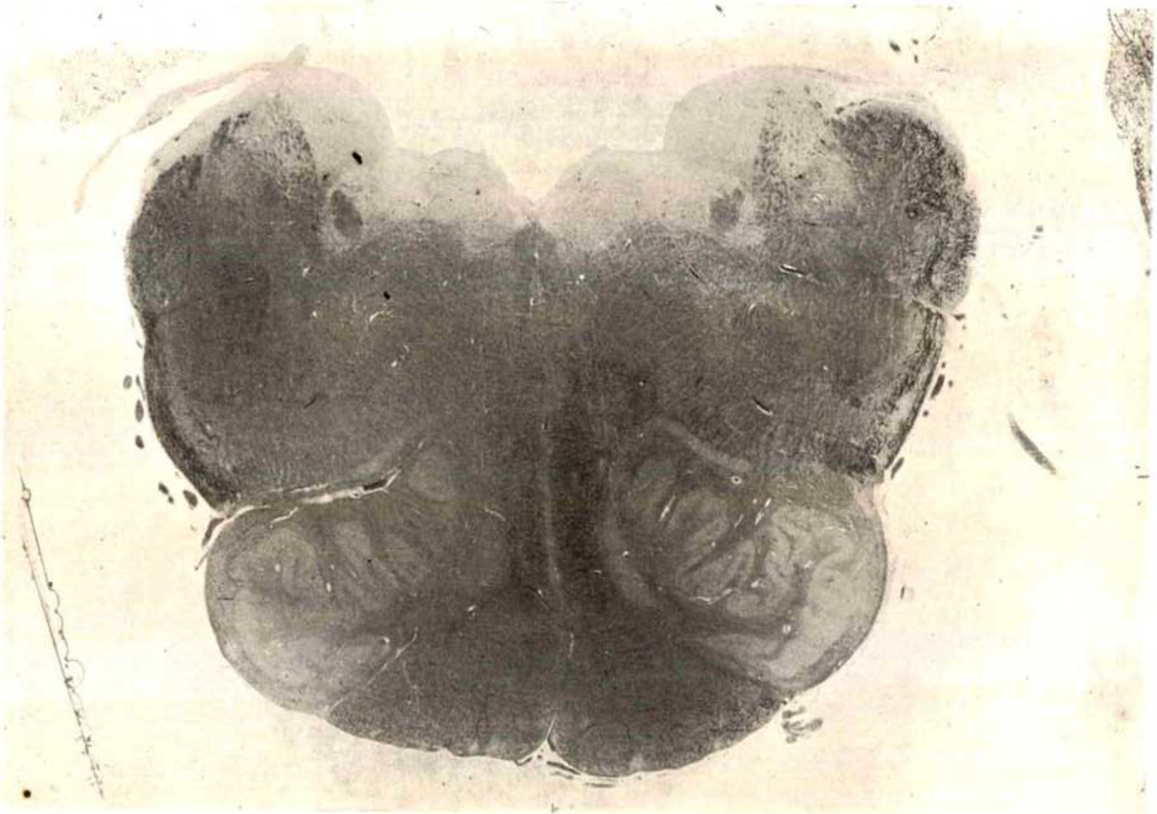

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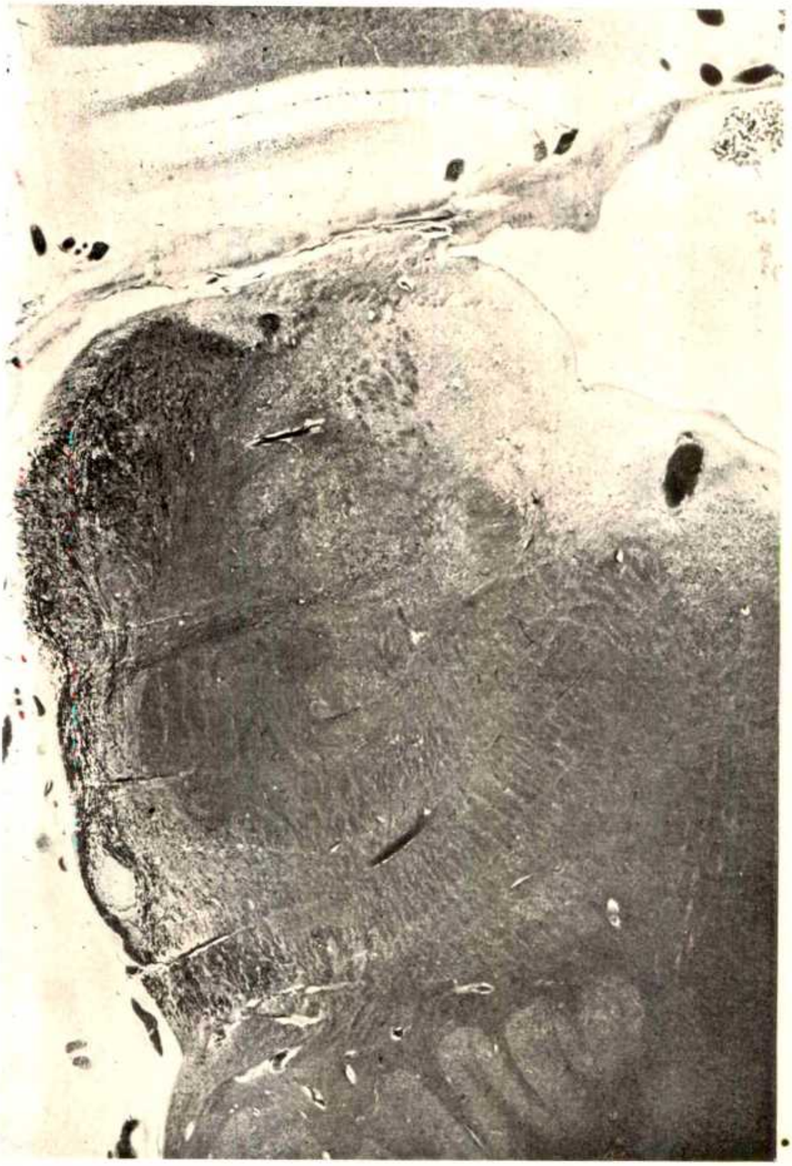


Dr. E. J. Worrall, *Phot.* 6 *Bale & Danielsson, Ltd., London.*

commences below in a plane close above the highest level of the pyramidal decussation. At the level of the upper end of the inferior olive the fibres of the dorsal plexus pass towards the central gray matter, and the plexus receives but few contributory fibres from the external arcuate fibres. The upper limit of this dorsal plexus is the highest level of the auditory striæ.

These fine plexuses, which in our preparations arise directly from the external arcuate fibres, were first noticed by Hoche in a case of transverse lesion of the spinal cord at the level of the seventh thoracic segment (*Archiv. für Psychiat.*, *Bd.* 28, 1896), and Hoche's drawings correspond very closely with our photographs.

It has been suggested, however, by Barker that these plexuses are the termini of spinal fibres ascending through the posterior column nuclei. As already stated, however, we have proved their origin to be from collaterals which arise from the external arcuate fibres.

But for the fact of the marked association in physiological activity between the vagus phenomena of vomiting and nausea and disturbances of equilibration it would be difficult to explain the close connection of the spino-cerebellar system with the nuclei of the vagus and glosso-pharyngeal nerves such as is provided by these plexuses.

(*c*) *Spino-vestibular fibres.*—As constituting a separate group of external arcuate fibres, we wish to draw attention to a well-marked bundle of medium-sized fibres which pass outside the thin layer of gray matter which covers the restiform body. These fibres curve round the restiform body and pass ventrally to the descending root of the vestibular nerve as well as towards the plexus just described as lying outside the fasciculus solitarius. Consequently there exists a spino-vestibular system which runs with the most dorsal direct spino-cerebellar fibres.

(2) *Gowers's Tract or the Fasciculus Spino-cerebellaris Ventralis. (Barker.)*

As the researches of Mott have shown that what was known as Gowers's or the ventro-lateral tract is really a complex of several systems, we have endeavoured to separate these systems, and, as stated above, regard them, at any rate, to be so far determined as to permit of their being enumerated as spino-cerebellar, spino-quadrigeminal, and spino-thalamic fibres, respectively.

It appears to us from examination of the present case that there is also another system included among the fibres of the ventro-lateral tract, and that is a small system of finer fibres which apparently terminate in the nucleus of of the lateral fillet.

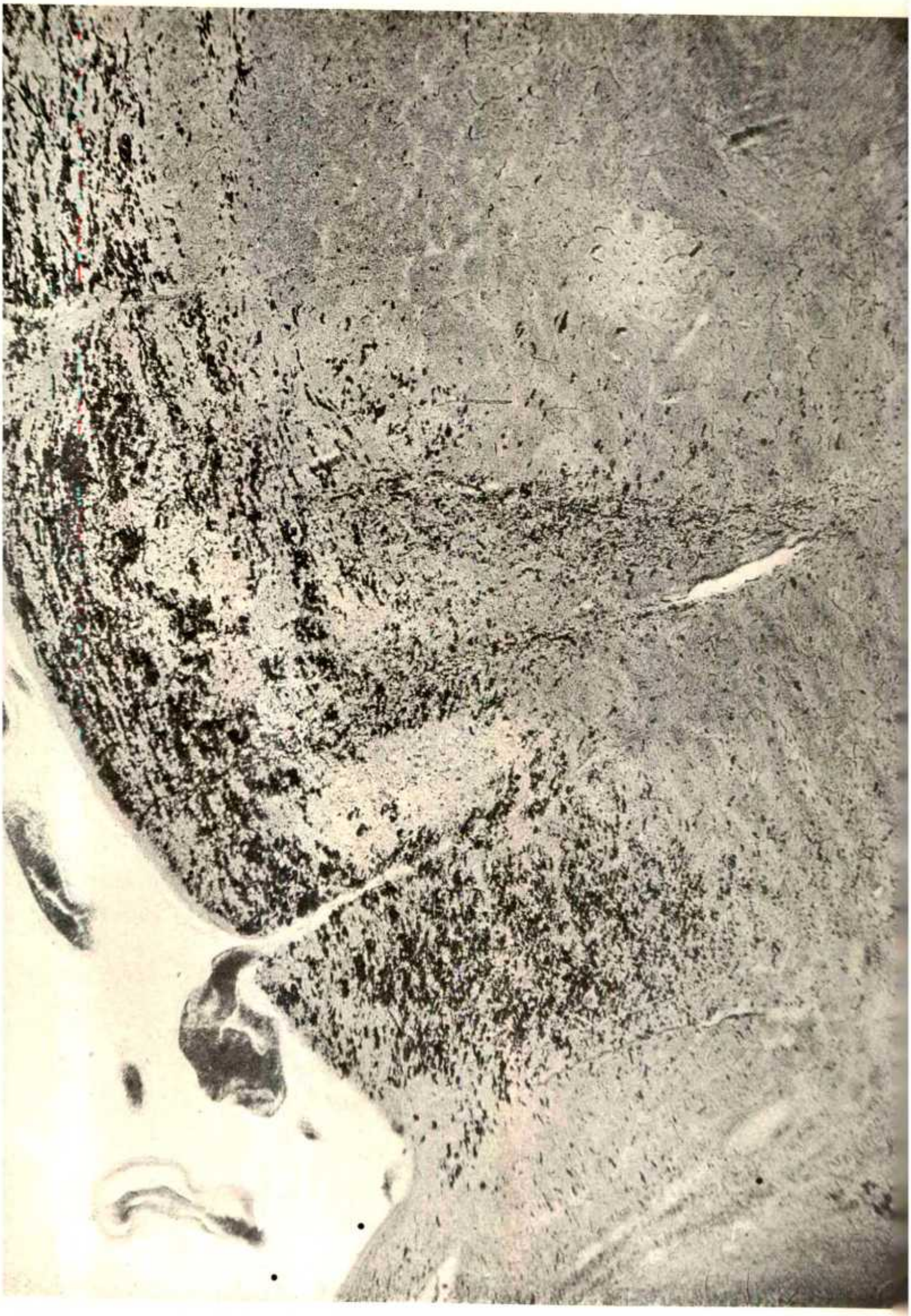
To deal with these points successively and systematically we venture to propose to make use of the expression Gowers's tract only in reference to the system of spino-cerebellar fibres.

We would point out prefatorily that the ventro-lateral field in the spinal cord shows a difference in the size of the fibres; that the outer fibres are rather coarser than the inner division, and further, that this appears to afford a basis for recognising the different tracts from each other. Thus in the higher sections up to (and including) the pulvinar region the coarser fibres pass by way of the superior peduncle to the cerebellum, but the finer fibres can be detected passing as spino-thalamic and as spino-quadrigeminal tracts.

The fibres of Gowers's tract proceeding to the cerebellum do so compactly as a thick bundle just outside the sheet of gray matter (continuous really with the central gray substance), which laps over and round the superior cerebellar peduncle. As the tract lies on the outer surface of the cerebellar peduncle it is intercalated as a layer between two other systems of fibres which are as yet undetermined, and on the highest point it is covered with a thin layer of sub-pial gray substance.

Owing to the fact that in the present case both tracts are completely degenerated, as well as the dorsal spino-cerebellar system, we are not able to add to the present knowledge of the termination of Gowers's tract in the anterior portion of the vermis.

At the same time we wish to draw attention to the following groups of fibres which bear a relation to the ventro-lateral spino-cerebellar system.



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Of these we would note first that fibres leave the tract, enter the velum medullare anterius and pass by way of the frenulum of the posterior corpora quadrigemina to the colliculus of either side.

The second group is much more important and difficult of analysis. Thus it is easy to trace fibres from one tract passing across the roof of the fourth ventricle to the opposite side, where they appear to descend in the sub-ependymal tissue towards the peduncle of the flocculus and the nuclei of the eighth nerve.

We must now discuss previous observations which bear on this group of fibres.

In the rhomboidal area bounded by Deiters's nucleus below, the bundles of the restiform body externally, the lateral wall of the fourth ventricle internally, and the brachium conjunctivum above, there have been for many years observed fibres extending upwards from the region of Deiters's nucleus and the embolus, and to the nucleus tecti in the cerebellum. These fibres are fine fibres, and have been variously described by different observers.

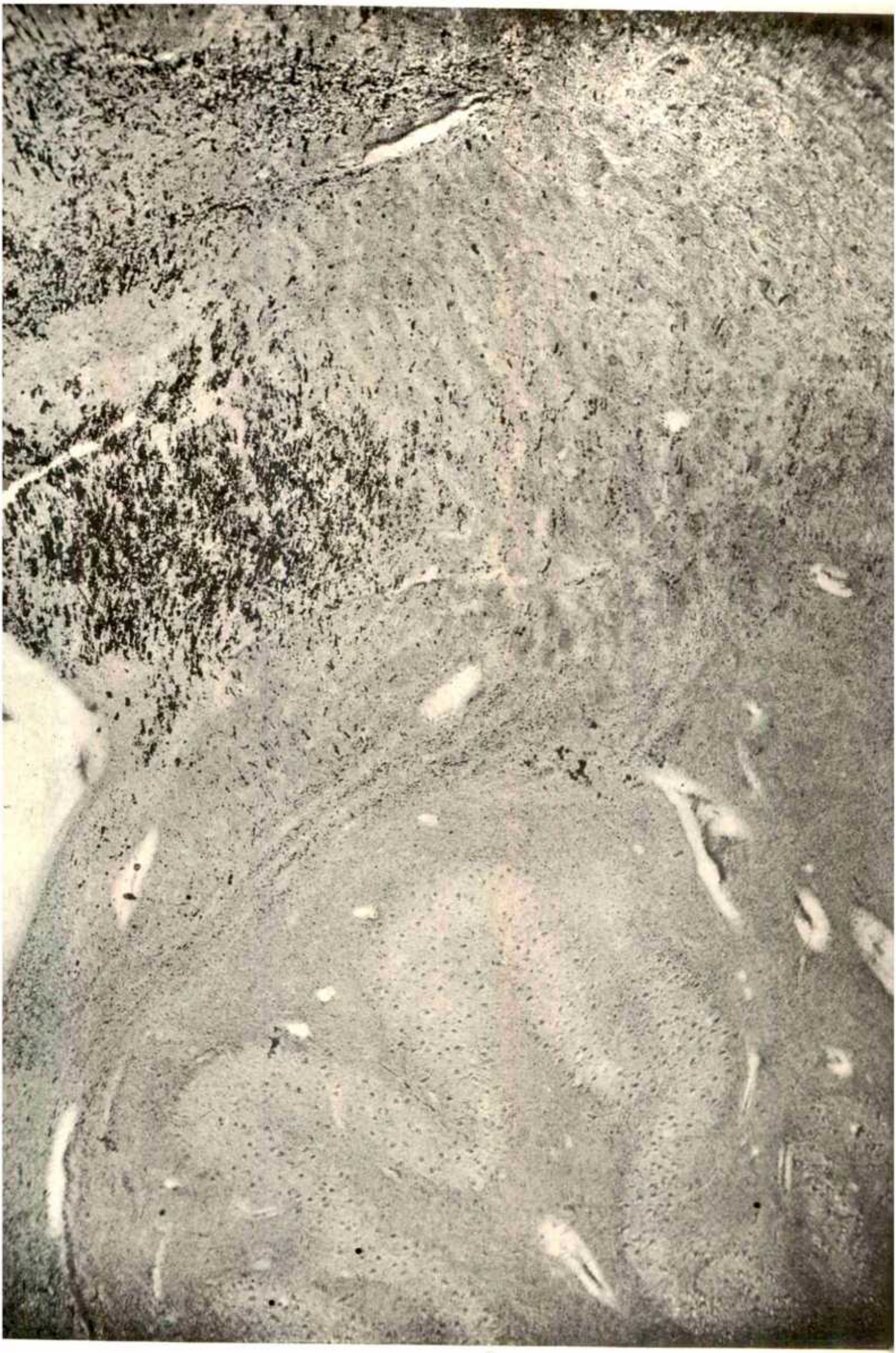
Thus Stilling (p. 175, table xv., *Stilling Disqu. de Struct. et Funct. Cerebri*) spoke of them as being the continuation of the column of the fasciculus cuneatus and the fasciculus gracilis. Bruce pointed out in 1892 that many of them came from the neighbourhood of the superior olive. Edinger, a few years later, showed that they were posteriorly apparently the continuations of various sensory cranial nerves, and he spoke of these bundles as a direct sensory cerebellar tract.

Kölliker describes them (p. 270, *Handbuch der Gewebelehre*) as a strong bundle passing dorsalwards on the mesial side of the restiform body. He traced them to the nucleus tecti, and partly through the embolus and globosus, finding that after decussating they terminated in the nucleus tecti of the opposite side. Thus Kölliker adopts the prevalent view that these fibres are such as leave the neighbourhood of Deiters's nucleus to go towards the cerebellum, and says that another origin to these fibres is not obvious. At the same time he makes the most important observation that he noticed in a human fœtus of the seventh to the eighth month the terminations of these fibres in the nucleus tecti to be already myelinated, whereas the "bundles of origin" were not, and thus contrasted very strongly with the medullated fibres of the nervus vestibuli and pedunculus cerebelli. This fact seems to us to render it at least improbable that the fibres ascend from Deiters's nucleus and the vestibular nerve into the cerebellum. In speaking of the region of these "bundles of origin," it should be noted that Bruce in 1892 showed the peduncle of the flocculus to spring from the area to which reference has been made. In the present case these fibres are degenerated, and have been described above as being possibly derivatives of Gowers's tract. In remarkable agreement with Kölliker's observation regarding myelination, we may again draw attention to the fact that in our case the lower ends of the fibres, where they are apparently connected with the upper part of Deiters's nucleus are distinctly branching and breaking up.

Our conclusions would be, therefore, that these fibres certainly come at any rate from the neighbourhood of the roof nucleus and pass down the side of the ventricle, to terminate in the upper part of Deiters's nucleus. Whether we are right in regarding them as being the continuation of the antero-lateral tract will have to be tested by subsequent observation, but in support of it may be mentioned that one of us has in the cat traced degenerated fibres of Gowers's tract across the roof of the fourth ventricle to the interval between the brachium conjunctivum and the lateral wall of the ventricle on the opposite side.

These same fibres are described by Miss Sabin in two papers, the most recent of which is that relating to her model of the medulla ("Contributions to the Science of Medicine, Dedicated to Wm. H. Welch, 1900," p. 990). In this connection she draws attention to two bundles of fibres connecting the vestibular nuclei with the cerebellum, which she calls inner and outer group respectively, of the fibres connecting the vestibular area with the cerebellar. Of these the mesial bundle passes into the roof.

The whole question of these fibres is a very difficult one



WORRALL, *Phot.*

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to solve, and with reference now to the question of Edinger's views regarding the direct connection of the vestibular nerve with the cerebellum, Ramon y Cajal (*Beitrag zm. Studium der Med. Oblongata, Deutsch. von Bresler*, Leipzig, 1896) includes in his fig. 18 fibres of the vestibular nerve, which, as they pass round the upper end of the spinal trigeminus root, bifurcate, the dorsal limb of the bifurcation running upwards, giving off collaterals to Deiters's nucleus, and terminating in the nucleus tecti. If this arrangement which is found in the mouse is confirmed for other animals and man, then some of these fine fibres must be taken as direct continuations of the vestibular nerve. Such a fact of course would not alter our view of the fibres observed in the present case, because such direct vestibular fibres would necessarily not be degenerated in our case.

Degeneration of the fleece fibres.—The connections of the corpus dentatum by means of the plexus surrounding it and known as the fleece (Stilling) are very important. In the present case numerous very fine degenerated fibres entered the plexus in large numbers on the dorsum of the corpus dentatum, but very few at the lower third of the ventral aspect of the nucleus.

As regards position in respect of the cerebellum as a whole it should be observed that the contributions the fleece receives from the restiform body enter its dorsal portion alone, so far as we were able to ascertain.

(3) *The Fasciculus Spino-tectalis vel Quadrigeminalis.*

This includes those fibres which terminate in the posterior and anterior colliculus respectively.

(*a*) *Fibres to the posterior colliculus.*—We have already stated that fibres leave the spino-cerebellar tract to enter the posterior colliculus by way of its frenulum. The main mass of fibres, however, to the posterior colliculus come towards its nucleus in association with the fibres which we find to end in the nucleus of the lateral fillet, and are running mesially to the latter. The spino-tectal bundles diverge beneath the nucleus of the posterior colliculus, forming a cup or funnel-shaped bed for the oval nucleus.

This description of the fibres beneath the ovoid nuclear centre of the posterior colliculus has been given by various anatomists for the fibres of the lateral fillet, and the spinal fibres are in the normal case not distinguishable from those of the lateral fillet, but are necessarily more caudal. In fact, the account given by Kölliker of the normal lateral fillet is exactly applicable to our spino-tectal (posterior) system.

(*b*) *Fibres to anterior colliculus.*—The termination of the spino-tectal system in the anterior corpus quadrigeminum is very definite. The fibres are at the most distal plane of the colliculus arranged in an oval bundle situated in the outer part of the deep medullary stratum, and therefore close beneath the posterior brachium. From this point the fibres ascending parallel to the longitudinal axis of the mesencephalon give off collaterals or turn off directly to run in the commissure between the corpora and in the deep medullary stratum. These give off collaterals at right angles, which enter the upper gray layers of the homolateral colliculus. Others pass across the middle line and enter the contralateral colliculus.

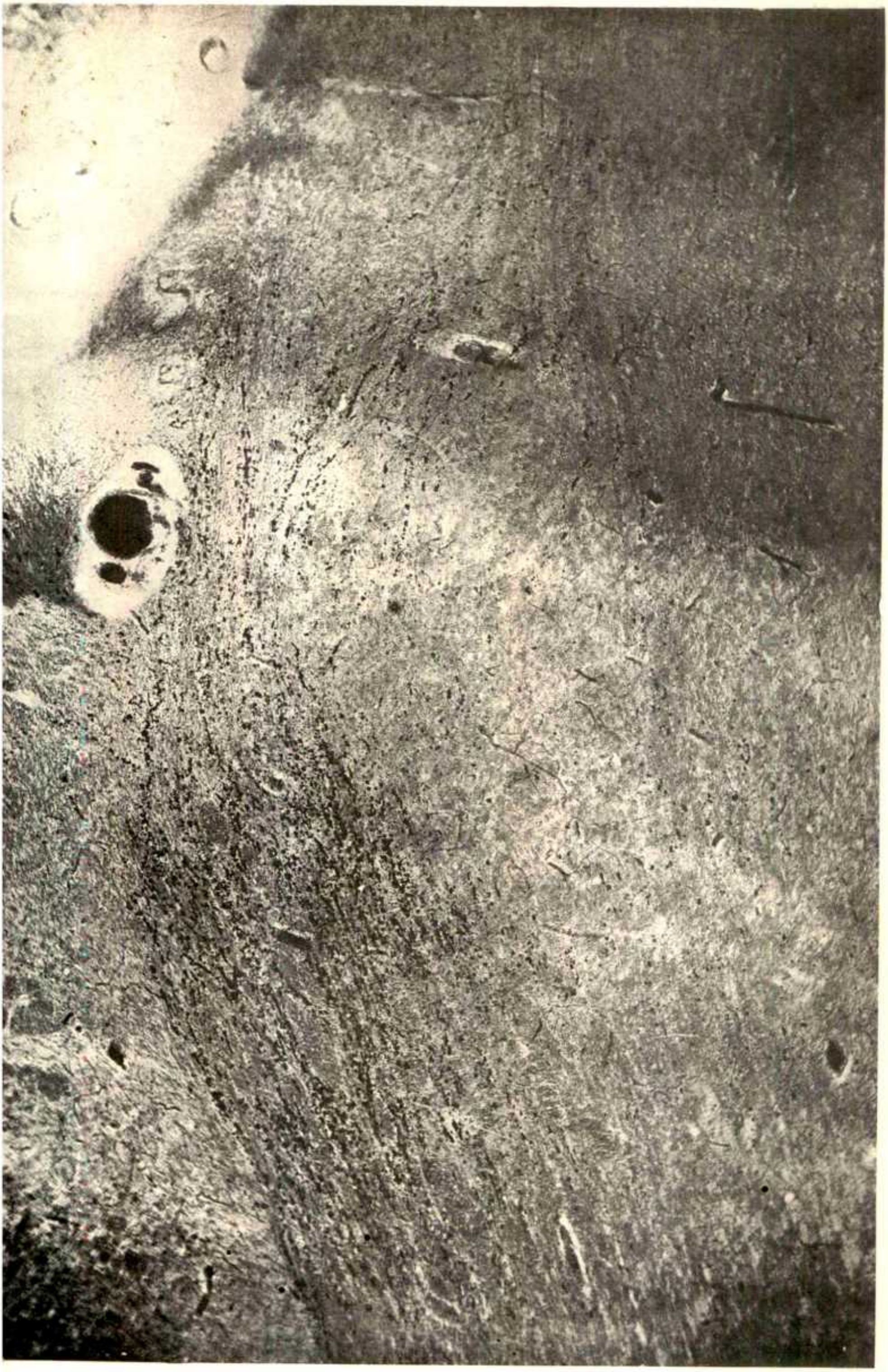
The gray layers of the corpus quadrigeminum thus receive the spinal fibres as follows: Those which end homolaterally pass into the outer part of the gray layers, and those which end contralaterally enter the inner portions of the gray layers.

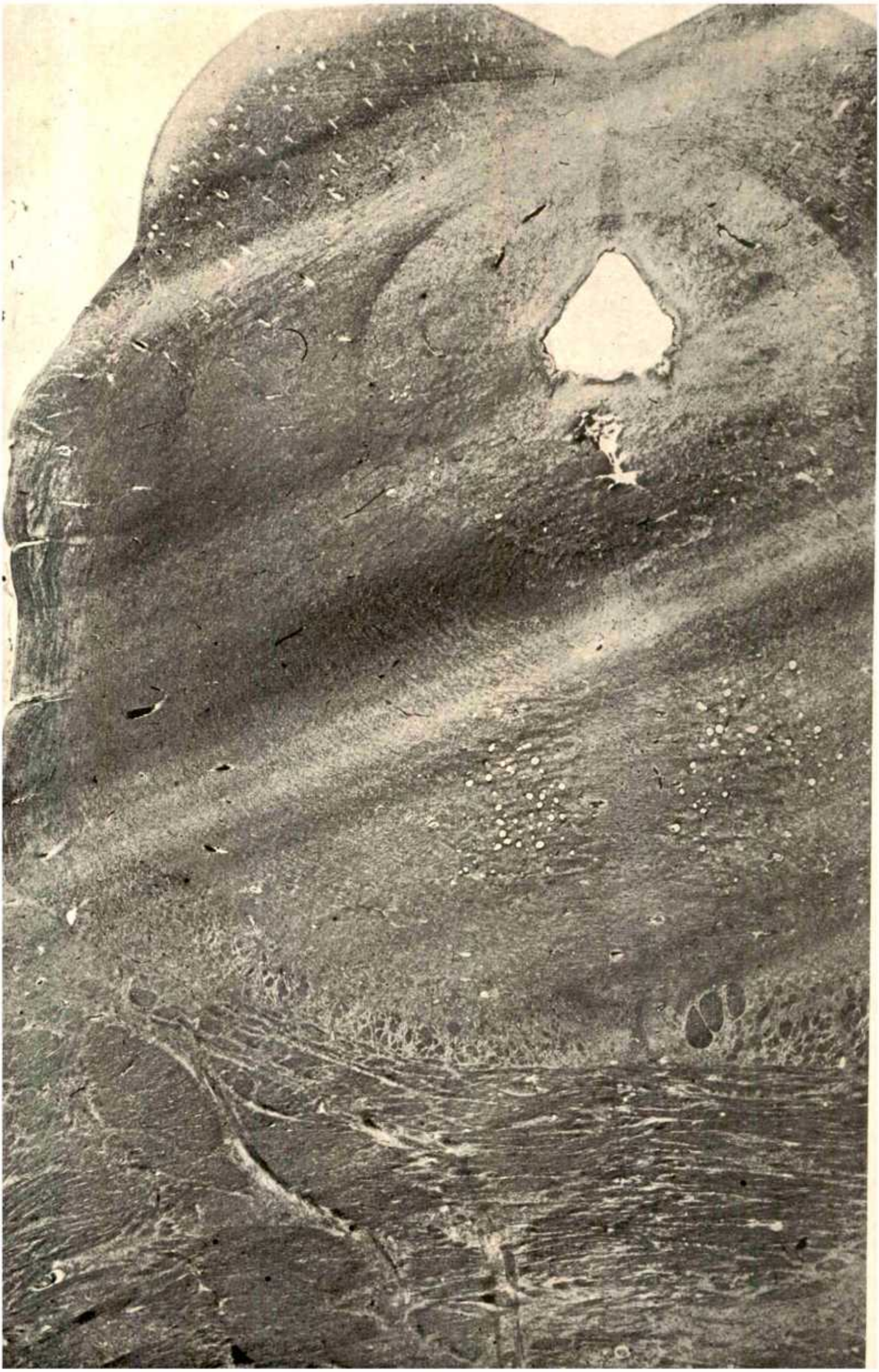
(4) *The Fasciculus Spino-thalamicus.*

After the ascending ventro-lateral tract has given off its contributions to the cerebellum and corpora quadrigemina as just detailed, the remnant of the fibres pass on to enter the thalamus.

In doing this the fibres spread out so that the bundle presents in transverse section a V-shaped figure. These fibres enter the pulvinar, and constitute, doubtless, a tract of considerable phylogenetic importance.

A few words may be added concerning degeneration in tracts other than those described.





(1) *Pyramidal system.*—A general and equal degeneration of coarse fibres was present in both pyramidal tracts, and in the spinal cord was spread over both the crossed tract and also the direct tract. The question whether this degeneration is possibly an ascending one has been raised by Dr. Purves Stewart in his case of injury of the cervical cord. In our case, however, sections across the parietal lobe showed not only extensive degeneration of large fibres descending (?) from the cortex, but also numerous medium-sized fibres passing into and across the corpus callosum.

Although no gross lesion of the cortex was discoverable in the osmic stained sections, we think that the above facts show that, probably as a result of concussion, the pyramidal corpuscles in the cortex suffered damage, and that their axones had degenerated in consequence. The patient in the present case undoubtedly received a severe shock, and, as mentioned above, suffered from a large scalp wound in the occipital region.

Pending further observations on this point it would perhaps be safer to regard the coarse degeneration of the pyramidal system as being of cortical origin and descending character.

Posterior longitudinal bundle system.—A few coarse fibres were traceable throughout the whole course of the posterior longitudinal bundle system. Thus in the anterior columns of the cord these fibres lay in front of the anterior commissure, and mingled ventrally with those of the direct pyramidal tract, occupying the mesial portion of the column. Higher up, at the level of the commencing decussatio lemniscorum, these same fibres lay close outside the fillet fibres. In their course up the medulla they kept close to the raphe, and finally in the mesencephalon formed the mesial border of the posterior longitudinal bundle. They began to disappear opposite the anterior corpus quadrigeminum, and at the commencement of the commissura mollis were reduced to a few units, appearing to end in the so-called nucleus of the posterior longitudinal bundle.

We have described these fibres as ascending in character because of their mode of termination and gradual disappearance headwards.

The "accessory fillet" (Schlesinger).—This is a tract which is in the main composed of medium-sized fibres. In the upper sections the fibres were compactly arranged and formed a tract, the fibres of which, passing downwards and outwards, became lost externally in the lateral fillet, and mesially in the tegmentum.

The tract first appears in the plane of the pulvinar. We believe with Redlich and Hoche that it is a downward efferent path of the pyramidal system, from which it obviously arises while traversing the pons.

The dorso-olivary fibres (von Solder).—At the commencement of the hinder pole of the olive von Solder's tract was visible as numerous scattered, fine, degenerate fibres and a few coarse ones, which latter were continuous with those from the anterior columns left after the decussation of the pyramids is established. The fine fibres are marked all along the dorsum of the olive, and limited, dorsally, partly by the ganglion cells of the formatio reticularis, while laterally they were continuous with the medium-sized fibres of the antero-lateral tract. Some fibres were scattered around the dorsal accessory olive, reaching outwards ventrally to the ventral nucleus of the funiculus lateralis and coursed round the surface of the bulb, just outside the capsule of the outer end of the inferior olive.

Some of the most mesial fibres passed into the inner side of the inferior olive.

As the fibres were traced upwards they almost all passed above the outer part of the fillet and entered the tegmentum. In this way they passed straight up to the thalamus and terminated by subdividing minutely in the ventral nucleus of the thalamus. von Solder traced the same fibres as far as the plane of the corpus geniculatum mesiale.

Posterior column system.—In the cord Goll's columns were equally degenerated on the two sides, the degeneration reaching only about three-fifths of the way along the median fissure. On the one side Burdach's column is affected in its median fourth, and for only two-thirds of its length posteriorly. The degenerated fibres could be traced upwards to the level of the pyramidal decussation, the nucleus gracilis

being there hardly marked, and at the same point the degenerated fibres became much finer.

At the lowest level of the bulb, where the corp. restiforme is distinctly formed, fine degenerated fibres, continuous with those of the posterior columns, form a delicate area of degeneration at the inner side of the restiform body, being separated from the degenerated spinal fibres by olivary fibres. These fine fibres join a fine meshwork of fibres in the dorsal part of the spino-vestibular root and nucleus, and appear to lose themselves in the gray matter just above the tuberculum acusticum, becoming finally lost beneath the ependyma as high up as the level of Deiters's nucleus.

The nuclei of the posterior columns show extensive degeneration, and from them a few fine degenerate fibres pass down as internal arcuate fibres into the fillet of each side. Similar fibres were first observed by Purves Stewart, but they disappear very soon from the fillet, and resemble fine collaterals rather than direct fibres.

Degeneration of the stria medullaris.—The fibres of the stria entering the ganglion habenulæ were in this instance degenerated, as also some coarse fibres in the posterior commissure. We were, however, unable to determine the cause or relations of these degenerations.

NOTE.—I desire to add to the above account the fact that the heavy labour of preparing the large series of preparations in the above case was executed by Dr. Thiele. —V. H.

THE OCCURRENCE OF OPTIC NEURITIS IN LESIONS OF THE SPINAL CORD. INJURY, TUMOUR, MYELITIS. (AN ACCOUNT OF TWELVE CASES AND ONE AUTOPSY.)

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AND

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CONSIDERING the rarity of recorded cases in which optic neuritis has been associated with lesions of the spinal cord other than cerebro-spinal meningitis, we venture to think that a series of twelve cases which have lately come under our observation are worthy of being recorded, especially since in one case a pathological investigation was made by one of us (J. T.). Our best thanks are due to Dr. Hughlings Jackson, Dr. Buzzard, Dr. Bastian, Sir William Gowers and Dr. Ferrier for permission to record cases under their care, and to Mr. Brudenell Carter and Mr. Marcus Gunn for permission to use their valuable notes upon these cases.

This paper contains :—

(1) An account of twelve cases.

The detailed clinical account of these cases will be found in the appendix at the end of this communication.

(2) A list of all the recorded cases we have been able to find in the literature of the subject.

(3) An analysis of all the cases of local lesions of the spinal cord which have been treated at the National Hospital during the years 1892-1900, with reference to the state of the optic discs.

Clinical Cases.

(*A detailed account of these cases will be found on p.* 541.)

Case 1.—*Tumour at the Third Cervical Segment.*

Female, aged 56 years. Slowly increasing spastic paraplegia and anæsthesia involving all four limbs. Marked weakness of muscles of neck. Well-marked double optic neuritis. Death two months later At the autopsy an extra-medullary intrathecal myxoma, about the size of a sparrow's egg, was found pressing into the cord at the level of the third cervical segment. There was no growth in the brain and no meningitis present

Case 2.—*Tumour at the Fifth Cervical Segment.*
(? + *Cerebral Tumour.*)

Male, aged 34 years. Syphilis at 27 years. Since 29 years periodic epileptic attacks, preceded by intense headache. Gradual onset of paraplegia for five months. Absolute flaccid paraplegia in all four limbs. Absolute anæsthesia everywhere below the fifth cervical area, except over lumbar 5 L., where all forms of sensibility were much impaired Much wasting and loss of faradic excitability in muscles supplied from fifth and sixth cervical segments. Well-marked double optic neuritis; swelling = 6½ D. left eye and 4½ D. right eye. Small hæmorrhages and effusions present in both discs. Six months later both discs presented well-marked post-neuritic atrophy, and vision was much impaired.

Case 3.—? *Tumour of Fifth Cervical Segment.*

Male, aged 48 years. Syphilis at 20. Gradually increasing spastic paraplegia of six months' duration, with shooting pains in the lower part of the neck. Left upper limb completely paralysed. The other limbs markedly paralysed. Complete analgesia and thermo-anæsthesia, with slight tactile impairment below the fourth cervical area. The edges of the optic discs were nowhere distinguishable.

There was exudation at the centre of the discs and along the vessels, with marked striation of the outer parts of both discs. There was no measurable swelling.

Case 4.—Spinal Caries.

Female, aged 14 years. Pain in cervical dorsal spine and gradually progressive spastic paraplegia of six months' duration. Paraplegia complete in three months; upper limit of anæsthesia one inch above the nipples. There was well-marked double optic neuritis, which increased markedly for the first fortnight of her stay in hospital, after which it gradually disappeared, leaving only slight blurring of the disc margin. There was an obsolete tubercle in one eye, and headache was present, but no vomiting. She was treated with rest and extension, and completely recovered from the paraplegia At the present time (nine years later) she is in the best of health.

Case 5.—Fracture Dislocation of Sixth Cervical Vertebra.

Male, aged 28 years. As the result of a fall on shoulder, pain and paralysis of both arms and gradually on-coming spastic paraplegia. Upper limit of anæsthesia at lower limit of eighth cervical area. Marked optic neuritis in left eye developed while he was under observation. A piece of bone pressing on the cervical segment was removed by laminectomy. The optic neuritis rapidly subsided, leaving atrophy of the left disc and vision = $\frac{6}{24}$ and J. 14. He completely recovered, and at the present time (nine years after) he enjoys good health, having been at work ever since he left hospital.

Case 6.—Acute Transverse Myelitis Seventh Dorsal Segment: Optic Discs Normal. A Second Attack Four Months Later in the Lower Cervical Region associated with Optic Neuritis.

Male, aged 24. In March, 1899, he awoke one morning to find himself paralysed and numb from the waist downwards. The usual symptoms of transverse myelitis were present. In July, 1899, while improving, slight optic neuritis was noticed. Four days later a rapid onset of paralysis of the hands, with complete anæsthesia below the third dorsal area, occurred. The neuritis increased rapidly, began to subside a fortnight later, and disappeared in four months, leaving obvious marginal blurring and connective tissue along the vessels. The symptoms of the cervical lesion improved greatly, those of the dorsal lesion did not improve.

Case 7.—Acute Myelitis Upper Dorsal Region. Evidence of past Optic Neuritis.

S. C., aged 36 years.—Rapid onset of paraplegia and anæsthesia, nine months before admission. Slight recovery of sensibility. In the right optic disc the edge was woolly and indistinct, the veins were distended and there was some connective tissue along the vessels. Some central filling and many fine vessels over the disc were present. There was no swelling. The left disc presented similar but less marked changes.

Case 8.—Acute Myelitis, upper limit—Sensory change—the Second Dorsal Area.

Male, aged 34 years. Paraplegia becoming complete in three weeks following an acute febrile illness, complete spastic paralysis (diaphragm excepted) below the fifth intercostal space. Upper extremities normal. Extreme hyperæsthesia round upper thorax (third to fifth rib). Thermo-anæsthesia as high as third intercostal space; pupils myotic.

Both optic discs were high coloured, and their edges were distinctly blurred though traceable, the retinal veins were overfull, appearances indicating an early stage of papillitis.

Case 9.—Acute Myelitis, Second Dorsal Segment.

Female, aged 22 years. Congenital syphilis. Acute onset of paraplegia and anæsthesia at twenty years old, followed by rapid failure of sight.

Both fundi showed advanced post-neuritic atrophy. Vision was reduced to light perception in the left eye, and to counting fingers at a distance of a foot with the left.

Case 10.—Lesion of Cervical Cord, (?) Hæmatomyelia.

Female, aged 15 years. Sudden loss of power and sensibility in arms, legs and trunk, accompanied by sharp pain between shoulders. No causal agent apparent.

Slight analgesia below fourth cervical area. Great atrophy and loss of faradic excitability in intrinsic hand muscles and in ulnar flexors. Double "main en griffe."

Absolute spastic paralysis of trunk and legs.

There was well marked optic neuritis in each eye, with several hæmorrhages near the centres of the discs.

The left pupil did not react to cocain, and the right only feebly.

Case 11.—Chronic "Myelitis," upper limit at the Fifth Cervical Segment. Post-neuritic atrophy of both optic discs.

Male, aged 41 years. Slow onset of spastic paraplegia for fifteen years. Slight anæsthesia to all forms below the fifth cervical root area. Both discs presented a blurred woolly indistinct margin with some connective tissue along the vessels, the latter being very small. Both were very pale and there was evidence of old exudation at the centre of each.

The condition of the discs was that typical of consecutive atrophy following a neuritis of moderate degree. It was first noticed by one of us (J. T.) two years before this patient's admission. No change had taken place in the appearance of the discs nor in the acuity of vision during this time.

Case 12.—(?) Myelitis, Upper Dorsal Region.

Male, aged 28 years.—Failure of vision associated with pain in dorsal spine, followed in a few weeks by onset of spastic paraplegia. Double optic neuritis leaving some post-neuritic atrophy at the end of three months. Left Argyll Robertson pupil. Upper limit of anæsthesia at the sixth dorsal level. Great improvement in symptoms at the end of three months.

The most striking feature of the above cases is that, notwithstanding the varied nature of the lesions, myelitis, compression, injury and probably hæmorrhage, the situation in all of them the cervical, or the upper dorsal regions of the cord. An analysis of the recorded cases (p. 552) shows a similar result, and if those cases which have been verified *post mortem* be taken alone, the lesions are found to have been confined to even a narrower limit in the downward direction, for we have been unable to find a recorded case in which the lesion did not extend higher than the third dorsal segment. In making this statement we exclude certain recorded cases in which the clinical and the pathological results were widely at variance, as for instance in Devic's case, where he repeatedly found the upper limit of anæsthesia above the nipple, and at necropsy the only lesion found was in the lumbar enlargement. It is difficult to explain the origin of the statement that has found its way into several recent text-books of medicine that optic neuritis in spinal-cord disease occurs in association with disseminated myelitis, and that in the large proportion of the cases the myelitis is confined to the dorso-lumbar region. Neither among the published cases nor among the records of the National Hospital for the past ten years have we been able to find any case of spinal-cord lesion associated with optic neuritis in which the clinical records or pathological results proved the cervical enlargement of the spinal cord to be uninvolved (except perhaps our own case 12), and conversely. We suggest, therefore, that the occurrence of optic neuritis in these cases is dependent in part upon the situation of the lesion.

The nature of the lesion in the above cases is various, the larger proportion being examples of transverse myelitis, which condition has been found in a large majority of the recorded cases. In three cases of disseminated myelitis which we have observed at the National Hospital, optic neuritis was not present, and in two only of the recorded cases was the existence of this lesion certain. A case of spinal tumour almost identical with the first of our series has been recently published by Nonne, and one of fracture dislocation of the vertebræ (C. 7, C. 8, D. 1, D. 2), resembling case 5, with a similar happy result by Firth. Several cases of spinal caries with slight changes in the optic discs are on record; Sir William Gowers states, however, that he is conversant with such cases, and that the changes are scarcely to be regarded as those characteristic of optic neuritis. We have not found optic neuritis of such degree as occurred in case 4 to have been recorded. The absolutely sudden onset of the spinal symptoms in case 10 rendered the diagnosis of hæmatomyelia very probable. Several cases in which this condition has been found *post mortem* will be found in the accompanying table.

In some of the recorded cases of myelitis associated with optic neuritis the disease has been narrowly limited, in others it has been of very wide extent, as, for example, in the case recorded by Mahokian and that by Schluster and Mendel, where the spinal cord was practically destroyed from the lower cervical to the sacral region. In about two-thirds of the cases of myelitis a history of syphilis has been obtained, and in relation with this fact it is noticeable that the incidence of the disease is chiefly in the third and fourth decades of life.

We have been unable to find among the records of the National Hospital any case in which optic neuritis was associated with syringomyelia. Neither was this sign present among a series of twenty-four fatal cases of subacute combined degeneration, notwithstanding the presence of severe anæmia in many of them, and that the disease involved the cervical region in the majority of them. In four, however, hæmorrhages were present in the retinæ.

We suggest, then, that the association of optic neuritis with a lesion of the spinal cord depends upon (1) the position of the lesion; (2) some peculiarity in the nature of the disturbance of the functions of the spinal cord produced by the lesion, for in the recorded cases the damage to the cervical enlargement, although in some cases extensive, has been partial only.

The occurrence of unilateral optic neuritis, or of neuritis of marked difference in its degree in the two eyes, in cases in which symptoms pointed to a great affection of one lateral half of the spinal cord as in case 5, and in two other recorded cases, is interesting in this connection. When the relative frequency of the occurrence of optic neuritis is considered, the records of the National Hospital show that even when instances of lesions of the cervical region alone are analysed collectively, optic neuritis occurs only in a small percentage of the cases.

In the larger number of the recorded cases, as in our series, the neuritis has not been of severe degree. Such neuritis increasing for a few weeks after its appearance usually has commenced to subside within two months of its onset whatever is the cause of the condition producing it, sometimes disappearing so completely as to leave hardly a trace of its former presence; it does not usually lead to an incapacitating degree of atrophy and loss of vision. In several recorded cases, as in cases 6 and 12, the neuritis has preceded the appearance of paraplegia by some days or weeks.

In not a few cases, however, the most intense optic neuritis with extensive hæmorrhages has occurred, and amaurosis shortly following the occurrence of the lesion, and subsequently progressing to complete and permanent blindness, has called the early attention of the patient and physician to the eyes.

It is interesting to note that in several recorded cases the sudden occurrence of reflex iridoplegia while the patient was daily observed has been noticed, sometimes preceding, sometimes succeeding, the occurrence of the neuritis. The Argyll Robertson pupil has been in some cases permanent and in some temporary only. Myosis and inequality of the pupil have also been observed. While some of our cases presented loss of the light reflex, this did not occur under our observation, neither, so far as we are aware, was the phenomenon transient.

We wish to bring forward the suggestion that, in addition to optic neuritis, headache, vomiting, or both, may occur in lesions of the cervical cord, especially in spinal tumours and pressure lesions. This association, which occurred in three of our cases, has been noticed also in other cases, and has led to the diagnosis of concomitant spinal and intracranial lesions. Yet subsequently the course of the disease or pathological investigation has rendered it probable or certain that such a double lesion had not existed. It is remarkable in this connection that, while cases of cerebral tumour presenting optic neuritis may run their course without either headache or vomiting, it is extremely rare for headache or vomiting to occur in those cases of intra-cranial growth which run their course without presenting optic neuritis.

We are unable to put forward any theory as to the causal relation of optic neuritis with lesions of the upper part of the spinal cord, but we hope at an early date to publish the results of experimental lesions upon the cervical cord in primates.

We may remark, however, that the most generally accepted explanation of such optic neuritis as being produced by two separate symmetrical foci of inflammation affecting the optic discs only, and occurring simultaneously with symptoms indicating a similar spinal lesion, is scarcely tenable in cases in which such optic neuritis is associated with local spinal tumours or injuries.

CONCLUSIONS.

(1) Optic neuritis of all degrees of severity may occur in connection with tumour, compression, myelitis or hæmorrhage affecting in some degree the upper part of the spinal cord.

(2) Headache and vomiting may be singly or together associated with neuritis from a local lesion of the cervical cord.

APPENDIX.

Case 1.—Mrs. M., aged 56, came under observation with the history that four years before she had begun to be troubled with difficulty in walking. This gradually became worse (although at times she could walk fairly well) until she became quite unable to use the lower limbs. To this, weakness of the arms was gradually superadded, and when she was first seen there was complete paralysis of all four limbs. There was also considerable although not complete impairment of the head movements. There was impairment of sensibility and the patient complained of a burning pain in the arms. There was also difficulty in passing water. One of us (J. T.) saw the patient at the suggestion of Sir W. R. Gowers, Mr. Victor Horsley and Mr. Marcus Gunn also saw her, and all four confirmed, besides the above symptoms, the presence of well-marked optic neuritis in both eyes.

The patient returned to her home, and about two months later died. The autopsy was performed about twenty-four hours after death by one of us (J. T.), kindly assisted by Dr. Henry Dobie and Dr. Herbert Dobie, of Chester. The spinal cord was examined and at the level of the third cervical root a small tumour about the size of a sparrow's egg was found outside the cord pressing on it. The most careful examination by all those who were present, of the brain and adjoining parts failed to reveal any intra-cranial growth. If any growth had been present it could scarcely have failed to be discovered.

Case 2.—H. J. T., aged 34 years, was admitted into the National Hospital under the care of Dr. Ferrier on April 14th, 1898, complaining of weakness of the legs and arms. The family history was good. He had acquired syphilis seven years before the onset of his illness.

His history was as follows: Five years before admission when in his usual good health he was suddenly seized with a fit in which he lost consciousness and was generally convulsed. A second similar fit occurred twenty minutes later, and a third attack in which the left side only was convulsed forty-five minutes later. After these attacks he is said to have had temporary weakness of the left arm and leg, He recovered perfectly in five months' time. A year later the fits re-appeared and have occurred since at irregular intervals. They were always preceded by several days of intense headache. Five months before admission he gradually became unable to stand, subsequently the hands were slowly affected and he became unable to write.

On admission he was a well developed man. Mental state fair, memory poor. He could only hear a watch on contact on either ear.

Taste and smell were normal, vision good with each eye. No contraction of visual fields.

There was well marked double optic neuritis in each eye. The edges of the discs were quite invisible. Swelling = 6½ D. left eye, and 4½ D. right eye. There were some particles of effusion and small hæmorrhages.

The cranial nerves were normal.

There was absolute anæsthesia from the upper limit of the fifth cervical segment downwards, except over the soles of the feet and lower part of anterior tibial region, where all forms of sensibility were much impaired. There was complete loss of sense of position in all four extremities.

There was absolute flaccid paralysis of both upper extremities, but the upper part of both trapezii, the sterno-mastoids, and the diaphragm acted normally, and he could shrug his shoulders. The intercostals were completely paralysed. Marked wasting of the deltoids and spinati, severe wasting of the other muscles of the upper extremities with loss of faradic excitability were present, and absolute paralysis of both lower extremities, frequent reflex flexor spasms, complete reflex incontinence. The knee-jerks were exaggerated. There was double foot clonus.

He remained seven months in hospital and gained some power in both arms and legs. The faradic excitability returned in those muscles from which it had been absent. He had little or no pain. One attack of general convulsions occurred while he was in hospital. When discharged on November 3, 1898, the optic discs presented well-marked signs of post-neuritic atrophy.

Case 3.—Thomas L., aged 48 years, was admitted into the National Hospital, under the care of Dr. Hughlings Jackson, on February 28, 1896, complaining of pain in the neck and weakness in the arms and legs. He had had syphilis twenty-eight years before admission. The pain dated from a fall on to the head from a height of twenty-six feet, three years before admission. Six months before admission the pain became much more intense, and his limbs gradually became weak. When admitted there was marked spastic paralyses of both legs, the feeblest movements alone being possible. Left worse than right, complete paralysis of left upper limb. All movements of right upper limb very feebly performed. There was complete analgesia and thermoanæsthesia to the upper limit of the fifth cervical root area. Sensation to touch was only very slightly impaired. There was incontinence of urine. The deep reflexes were much exaggerated. The optic discs presented no measurable swelling; the edges were nowhere distinguishable. There was some central exudation and exudation along the vessels. Stricture was present on the outer parts of both discs. The pupils were unequal, but reacted normally to light and to cocain. The other cranial nerves were normal.

Case 4.—E. B., aged 14 years, was admitted into the National Hospital, under the care of Dr. Buzzard, complaining of difficulty in walking and pain in the upper part of the back, radiating round the chest. There was a family history of tubercular disease. The history of the illness was as follows: Six months before admission she noticed a pain in the upper part of the back which ran round the top of the chest, and a fortnight later the knees began to feel numb, and the legs to become stiff in walking.

Three months before admission, on waking one morning, she found that she could not move either leg and had no feeling in them. She had not improved since this time. She had never had headache or vomiting during her illness.

On admission she was a well nourished, healthy looking girl. There was a marked angular curvature involving the third, fourth and fifth dorsal spines. The prominence was tender on pressure and there was pain in any movement of the spine. The special senses, cranial nerves and upper extremities were quite normal.

There was complete spastic paralysis of the lower extremities.

Absolute anæsthesia to all forms of sensibility below Poupart's ligament and the iliac crest. All forms of sensibility much impaired as high as level of the umbilicus. Slight blunting of sensibility as high as an inch above the nipples.

The optic discs were examined by Mr. Brudenell Carter, who found double optic neuritis more marked in the left than in the right eye.

On August 3 the neuritis was found to be much increased by Mr. Marcus Gunn. She had complained several times of frontal headache.

On September 23 the optic neuritis had almost disappeared. Voluntary power was returning in the legs, and sensibility had been completely regained. On March 5, 1889, she left the hospital completely recovered except that she occasionally suffered with slight headache. The discs showed marked signs of past neuritis.

She was treated with rest and extension to the spine. Counter-irritation was applied to the back by means of the actual cautery. Cod-liver oil and iodide of iron were the drugs employed.

Case 5.—W. B., a gilder, aged 28 years, was admitted into the National Hospital, under the care of Dr. Buzzard, on September 27, 1892, having four months before fallen off his van into the road, striking the back of his right shoulder. He was under the influence of alcohol at the time, and was able to get up and walk home, a distance of 300 yards. He noticed nothing wrong till next morning, when, on waking, he was unable to move the right arm on account of severe pain. A fortnight later the pain had become more severe and both arms became powerless. During the next two months he slowly lost control of the legs and sphincters.

When admitted he was a well nourished, intelligent man. A watch was heard only on contact with left ear, the membrana tympani having been destroyed by otitis media.

Vision: right eye $\frac{6}{6}$ and J. 4; left eye $\frac{6}{24}$ and J. 14. The right optic disc was normal and there were about three dioptres of general hypermetropia. The left optic disc was much injected, outline concealed, veins large and distended.

The right pupil was persistently larger than the left, and both reacted normally, otherwise the cranial nerves were normal.

Motor system.—He was unable to move or turn in bed. In the lower extremities slight power of extension of knee and ankle were present. There was marked clasp-knife rigidity, and painful recurring reflex flexor spasm. The abdominal walls were rigid.

In the right upper extremity he had lost all power of flexing or extending the fingers and wrist. The biceps, deltoid and supinators and pronators of the forearms acted well. The triceps was a little weak.

In the left hand all intrinsic movements of the hand and fingers were lost, but the movements of the wrist were good. The triceps was slightly weak and the other muscles acted normally.

The intrinsic hand-muscles on each side were wasted. There was no change in the electric excitability either to faradism or galvanism. The right arm as a whole was smaller than the left.

Sensibility.—He complained of pain, persistent and increased on movement, in the region of the sixth and seventh cervical spines. These spines were tender to deep pressure. Sensation to touch was practically perfect all over the body. Sensibility to pain was lost or greatly impaired everywhere below the eighth cervical root area. Sensibility to cold was normal, that to heat being slightly impaired over the affected area. There were some trophic changes on the skin of each hand, and profuse perspiration over both palms. The knee jerks were much exaggerated and there was double foot clonus. He was unable voluntarily to start the acts of micturition and defæcation. There was no spinal deformity.

On October 24 Mr. Horsley performed laminectomy opposite the sixth cervical vertebra, the leptomeninges were adherent to the theca, and a transverse ridge of bone projected backwards from the body of the vertebra and pressed upon the cord.

On October 26 the left optic disc was blurred and its outline was invisible. Surface seen with + 5 D. The right disc was injected and its edge was deficient in sharpness.

On November 3 there was marked improvement in movement in the arms and legs. He had had no pain since the operation. He was able to walk on January 29, and was completely recovered on September 19. The right optic disc at this time was normal; the left disc showed partial atrophy and white radiating striæ; its edge being invisible. There was no swelling. He has remained well since.

Case 6.—S. S., an engineer, aged 24 years, was admitted into the National Hospital, under the care of Dr. Ferrier, on July 7, 1899, complaining of loss of power and of feeling below the waist and incontinence of urine. Family history unimportant. Four years previously he had venereal disease with a non-indurated sore, and two years later he had an ulcerated throat, which rapidly healed under the influence of black wash.

In March, 1899, after having felt a curious cold feeling round his loins and some slight weakness of the legs for a week, he awoke one morning to find that he could not move either leg, and he was numb from the waist down. The sphincters were incontinent.

Six weeks later he was admitted into King's College Hospital under Dr. Ferrier. The knee jerks were noticed to be absent and the paralysis complete. A month later the right knee-jerk returned. The optic discs were normal at this time.

On admission there were no symptoms of intracranial disease. The mental state, special senses, and cranial nerves were normal.

Both optic discs were very red and their edges were blurred. There was radial white striation all over the discs. The swelling = 2 D. The arteries were pressing very much upon the veins.

The pupils were equal and re-acted normally.

There was absolute flaccid palsy of both legs, but the electrical excitability of the muscles was normal. The rectus abdominis did not contract below a point two inches above the umbilicus in the attempt to sit up (seventh dorsal). There was greatly diminished sensibility to touch below the seventh dorsal root distribution and almost complete analgesia, thermanæsthesia, and loss of sense of position below that area. There was absolute anæsthesia, incontinence of urine and fæces, and a deep sacral bedsore.

The plantar reflexes were extensor responses. The knee jerks could with difficulty be obtained with double reinforcement. The wrist and elbow jerks were normal.

On July 29 he complained of pain down both arms, and the grasps were found to be very weak. The arms were generally paretic, but the scapula-thoracic and humero-thoracic muscles were normal, as was also the deltoid.

Relative anæsthesia and analgesia were at this time present from the nipple downward, and there was some hyperæsthesia over the inner aspects of both upper limbs.

Three days later there was complete anæsthesia and analgesia to a line round the thorax 1½ inches above the nipples.

On November 4 the upper extremities had slowly and completely recovered power. The anæsthesia was still almost complete below the nipples. The swelling of the optic discs had subsided, their edges were woolly and indistinct. There were obvious signs of old exudation on the surface of both discs.

Case 7.—Samuel C., aged 36 years, was admitted into the National Hospital under the care of Dr. Ferrier on May 27, 1896, complaining of paralyses of the legs, headache and dysuresis. He denied venereal disease. He had malaria in America seven years before admission. Nine months before admission he was seized with dull pain in the back and numbness in the legs as he was walking. Weakness in the legs and difficulty in passing water followed a few hours later, and at the end of forty-eight hours the legs were completely paralysed, and he was numb up to his waist.

Three weeks later he began to recover sensation in the legs. At the time of onset of his illness he had much headache and general malaise. When admitted there was complete spastic paraplegia, and sensibility to all forms was diminished below the fourth dorsal root level. The deep reflexes were much exaggerated and there was double foot clonus. The pupils reacted normally, and the cranial nerves, other than the optic, presented no peculiarity.

Optic Discs.—Right, edge indistinct and woolly; veins distended, some connective tissue along vessels; some central filling and many fine vessels over disc; no swelling. Left similar, but less markedly abnormal.

Case 8.—W. J. E., a butcher, aged 34 years, was admitted into the National Hospital under the care of Dr. Bastian on August 23, 1892, complaining of loss of power and sensibility in the legs and incontinence. His health, previous to this illness, had been good. He had had five attacks of gonorrhœa. Five years before admission he had symptoms which were presumably those of syphilitic infection. In March, 1892, he was laid up with an illness accompanied by pain in the back and shivering. He was acutely ill for a fortnight, at the end of which time he noticed that he was losing power in the left leg. In three weeks he completely lost power in both legs, and in the lower part of the trunk.

On admission he was completely paralysed below the fifth intercostal space, the diaphragm, which acted well, being excepted. The lower extremities were rigid, and there were at times reflex flexor spasms. The upper extremities were normal. There was a band of extreme hyperæsthesia extending round the thorax, its upper limit being the third intercostal space in front and its lower limit the lower edge of the fifth rib.

There was complete loss of tactual sensibility over the fourth and fifth lumbar and all the sacral areas of the left side, and partial loss over the same areas of the right side. There was analgesia over the right half of the body and right leg, limited sharply above by the sixth rib in front and eighth dorsal spine behind and by the mid-line of the body.

Thermo-anæsthesia extended as high as the third intercostal space. There was complete loss of sense of position and of passive movement in the lower extremities.

Knee-jerks exaggerated. Double foot clonus.

Complete loss of control of sphincters.

Pupils small and equal and re-acted to light and during accommodation.

Both optic discs full coloured and edges decidedly blurred though still traceable, the retinal veins were over full, probably an early stage of papillitis. He suffered occasionally with headache and vomiting.

On December 6, 1892, the optic discs were normal.

Case 9.—G. M., aged 22 years, was admitted into the National Hospital, under the care of Dr. Bastian, in February, 1899, suffering with complete loss of power in the legs and incontinence of sphincters.

When 6 weeks old she developed signs of congenital syphilis. When 7 years old she suffered with interstitial keratitis and her sight became in consequence somewhat impaired. When 13 years of age some lateral curvature of the spine was noticed. At 14 she had chorea. When 20 years old in the course of a week she lost the feeling and use in her legs and became completely incontinent. Some weeks after her sight began to fail and her memory became much impaired. She had not improved up to the time of her admission. When admitted she could perceive light with the left eye and could only count fingers with the right. Both fundi shewed advanced post-neuritic atrophy. Leucomata were present in both cornea. Old iritic adhesions were present in each eye and the pupils did not respond to light, the right alone contracting slightly on convergence. The other cranial nerves were normal.

There was absolute spastic paraplegia below the chest, and complete anæsthesia to all forms below the ninth dorsal level. Marked impairment to pain and touch was present up to and including the second dorsal root area.

Case 10.—A. S., a schoolgirl, aged 15 years, was admitted into the National Hospital, under the care of Dr. Hughlings Jackson, on October 5, 1898. The family history was good and she had always been healthy. A fortnight before the onset of this illness she fell off a stile and hurt her back slightly. Fifteen months before admission and a fortnight after the fall on her back, on getting into bed one night she felt a sudden sharp pain between the shoulders and "went out stiff" in her arms and legs. She completely lost power in the arms and legs at once and could not feel below the upper part of the chest; there was complete loss of control over the sphincters.

Up to the time of her admission the arms improved somewhat, but the legs remained, completely powerless. Flexor spasm and bed sores troubled her greatly.

On admission there was slight analgesia over the whole body below the fourth cervical area.

Great atrophy of all intrinsic hand muscles and of ulnar flexors. Other muscles of the upper extremity normal. Double "main en griffe."

Intercostals completely paralysed. Diaphragm acted well. Absolute spastic paraplegia in trunk and legs. Double foot clonus.

Mr. Gunn examined the eyes and made the following note:—Well marked optic neuritis in either eye. Swellings = 3 D. Edges of discs quite blurred. Several small hæmorrhages near the disc in both eyes. Pupils equal and normal, but the left did not re-act to cocain and the right only re-acted slightly.

Case 11.—*Post-neuritic optic atrophy in chronic "myelitis," upper limit of lesion in the fifth cervical segment.*—J. C., aged 41 years, a police constable, was admitted into the National Hospital under the care of Dr. Bastian on June 25, 1895, complaining of weakness of the legs. There was a history of venereal disease (nature uncertain), nineteen years before admission. He had noticed gradually increasing weakness in the legs for fifteen years and had been unable to work for seven years. When admitted his condition was one of marked spastic paraplegia involving both arms and legs. He could still walk with a stick. The pupils reacted normally and there was abnormality in the cranial nerves other than the optic. Vision R.E. $\frac{6}{36}$, L.E. $\frac{6}{24}$, J. 4 in both eyes.

Both discs presented a blurred, woolly, indistinct margin, with some connective tissue along the vessels, which were very small. Both discs were very pale, and there was evidence of old exudation in the centre of each.

The upper extremities were weak and rigid. There was slight anæsthesia to all forms below the fifth cervical area. Some rigidity was present in all four limbs. There was no local wasting of muscles. All the deep reflexes were exaggerated and there was double foot clonus.

The nature of the lesion in this case was uncertain. The upper limit of its extent in the spinal cord was definitely the fifth cervical segment. The condition of the optic discs was that typical of consecutive atrophy following a neuritis of moderate degree. This atrophy was first seen by Dr. James Taylor two years before admission, and the condition of the discs and vision had not changed during the period he was under observation. He had never at any time suffered with headache, vomiting, or cerebral symptoms.

Case 12.—E. K., a compositor, aged 28 years, was admitted into the National Hospital under the care of Dr. Hughlings Jackson on June 18, 1900. His health had been very good before the onset of this illness. He had not had symptoms of lead poisoning and he denied venereal disease. At Easter, 1900, he suffered with lumbago for four days and at the end of the same week he noticed that his sight was failing and that his legs were numb and weak. He went to Moorfields and came under the care of Mr. Morton, who discovered that optic neuritis was present. He was admitted, and his sight gradually improved, but the legs became more and more weak and the numbness gradually involved the trunk from below up, and he developed dysuresis. He had never had headache during the illness, but had vomited several times during the first few weeks. A girdle sensation of tightness had been present above the waist for some weeks.

When admitted to Queen Square, vision was Rt. eye $\frac{6}{6}$ and J. 1; left eye $\frac{6}{9}$ and J. 14. Left field concentrically contracted. Both discs showed post-neuritic atrophy L > R.

The left pupil did not re-act to light: otherwise the cranial nerves were normal. Slight weakness and marked tremor on movement in the intrinsic muscles of both hands. Muscles of the abdominal wall rigid. The lower extremities were weak and spastic, the right much more so than the left. The knee jerks were exaggerated and ankle clonus was present. He was unable to walk.

There was anæsthesia to light touches everywhere below the level of the ensiform process. The deep reflexes were increased in the lower extremities, and the plantar reflexes were extensor in type. He had difficulty in starting the act of micturition. He left the hospital on August 29, able to walk well though still showing a spastic-ataxic gait; the anæsthesia and dysuresis had disappeared.

Table of 240 Cases of Local Disease of the Spinal Cord, from the Records of the National Hospital, 1893—1899 inclusive.

Nine of the Writers' Cases are here included.

The figures in dark type refer to cases in which definite papillitis or post-neuritic atrophy has been observed; those in ordinary type to cases in which the optic discs presented no changes. All cases in which albuminuria, lead poisoning, or extensive vascular degeneration was present have been excluded. The spinal segment referred to denotes the approximate upper limit of the lesion.

In 86 cases either there was no record of the condition of the optic discs or the records were too scanty to determine even approximately the limits of the lesion.

The remaining 154 cases are here tabulated.—

	C.5.	C.6.	C.7.	C 8.	D.1.	D.2.	D 3	D.4.	D 5.	D.6.	D.7.	D 8.	D 9.	D 10.	D.11.	D.12.	L.1.	L 2.	L.3.	L.4.	L.5.	
Myelitis	2, **2**	**1**	1, **1**	1, **2**	2, **1**	1	0	4	3	2, **1**	4	2	12	9	8	4	7	2	1	—	—	= 60, **8** = 68
New growth	1	1, **1**	1, **1**	2	1	**1**	0	0	0	0	2	1	1	0	1	0	2	2	1	0	1	= 17, **3** = 20
Hæmatomyelia .. .	1, **1**	0	**1**	0	1	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	= 4, **2** = 6
Fracture dislocation ..	0	0	**1**	1	**1**	—	—	—	—	1	—	—	1	1	—	1	2	1	1	—	—	= 9, **2** = 11
Pachymeningitis ..	**1**	—	—	—	**1**	—	—	1	—	2	—	—	—	—	—	—	—	—	—	—	—	= 3, **2** = 5
Spinal caries	**1**	0	1, **1**	2	4	1	2	4	4	4	4	3	3	3	4	1	1	1	—	—	—	= 42, **2** = 44
Optic discs normal ..	4	1	3	6	8	2	2	9	7	9	10	6	17	13	9	6	13	6	3	0	1	= 135
Neuritis present.. ..	**5**	**2**	**5**	**2**	**3**	**1**	0	0	0	**1**	0	0	0	0	0	0	0	0	0	0	0	= **19**

	With optic neuritis.	Without.
Lesions affecting cervical enlargement	18 ..	24
" " dorsal region	1 ..	88
" " lumbo-sacral region	0 ..	23

TABLE OF PUBLISHED CASES.

The numbers in the sixth column refer to the upper limit of the lesion as verified or deducible from the symptoms. In several cases the ophthalmic signs have been recorded in great detail, but the spinal signs with so little, that it has been impossible to state definitely the position of the lesion.

Author	Reference.	Sex.	Age.	Lesion	Situation.		
Abadie	*Bull. Soc. Chir.*, Jan. 12, 1876						
Achard and Guinon	*Arch. Med. Exp.*, 1889, p 696	M.	30	Diffuse myelitis	Cervico-dorsal + at D. 8	O. N. preceded	+
Allbutt	"Gower's Med. Opthal.," 3rd ed., p. 189	—	—	Myelitis	Upper dorsal		
Bielschowsky	*Neur. Centralbl.* 1901, p. 457	M.	43	,,	C. 8—D. 1		
Bull	*Amer. J. Med. Sc.*, July, 1875						
Chauvel	*Bull. Soc. Chir*, 1880, p 512	M.	23	,,	D. 1		
Chisholm	*Arch. f. Ophth.*, xi., No. 2, June, 1882	M.	28	? Hæmatomyelia	D. 1—2	O. N. preceded	
Devic	*Cong Franç. de Med.*, 1894, p. 434	F.	45	Myelitis	Upper dorsal		+
Drake-Brockman	*B. M. J.*, 1892, pt 2, p. 77	M.	19	,,	Cervical	Vomiting	
Dreschfield	*Lancet*, 1882, p. 8052	F.	38	Softening	Cervical enlargement 1½ ins.		+
	B. M. J., 1894, p. 474	M.	41	Myelitis	D. 1		+
		M.	23	Disseminate myelitis	Throughout cord		+
Elschnig	*Arch. f. Augenheilkunde*, xxvi.	M.	26	Myelitis	Mid-dorsal ?	Argyll Robertson pupils	
		M.	22	,,	?	,,	
		M.	38	,,	?	,,	
		F.	31	,,	?		
Erb and Steffan	*Arch. f. Psych.*, x., p. 146	M.	50	,,	Upper dorsal		
Eskridge	*J. N. and M. Dis*, N.Y., 1890	M.	49	,,	C. 8 and D. 1		+
Firth	*Practitioner*, 1886	M.	23	Fracture dislocation	C. 6, 7. 8; D. 1		
Gault	Lyon, 1894						
Gowers	*Med. Ophthalmoscopy*						
Hoffmann	*Neur. Centralbl.*, 1896, S. 671	M.	48	Myelitis	Cervical enlargement		
		F.	56	,,	Upper dorsal		
Katz	*Arch. für Ophth.*, xlii.	F.	56	,,	Cervical enlargement		+
Knapp	"Tagebl. der 58 Versamml. deut. Naturf. und Aertze." Strasburg, 1885, p. 489	M.	32	,,	D. 1		
Mahokian	*Inaug. Dissert*, Berlin; reference with case, No. 24	M.	28	,,	C. 8—S. 4		+

[illegible]	[illegible] 1894						
Nonne	*Arch. f. Psych.*, lxxxiii., f. 2, 1900	F.	15	Sarcoma	C. 2		+
Noyes	*Arch. f. Ophth.*, ix., 1880, pt. 11, p. 129 *Arch. f. Augenheilkunde*, 1881	M.	25	No details			
Rieger and Forster	*Arch. f. Ophth.*, xxvii, 3, p. 109	—	—	Myelitis	No details		
Ripamotti	*Atti dell'assoc. Med. Lomb.*, Milan, 1896						
Rumpf	*Deut. Med. Woch.*, 1881, p. 442	F.	32	,,	Details deficient		
Schatz	*Deut. Med. Woch.*, 1893, p. 615	M.	19	,,	Lower cervical		
Schluster and Mendel	*Neur. centralbl.*, 1899, p. 1088	M.	23	Diffuse myelitis	Complete destruction of cord below C. 8		+
				,, ,,	C. 2, &c.		+
				Dissem. ,,	Largest focus at D. 2		+
Schlüter, F.	"Ueher opt. neur.," *Marg. Dis.*, Berl., 1882	M.	49	Myelitis	?		
Sequin	*J. Neur. Ment. Dis.*, N.Y., April, 1880	M.	35	,,	Upper dorsal		+
Sharkey	*Med. Times*, 1884, i., p. 812	F.	19	Softening	Cervical enlargement		+
Sharkey and Lawford	BRAIN, viii., 133 (Abstract)	F.	17	Myelitis	Cervical region, upper and lower	O. N. preceded	+
Taylor, F.	*Guy's Hosp. Reports*, 1896, 53, p. 45	F.	33	,,	Cervical		
Taylor, James	*Allchin's Medicine*, vol. iii., p. 894	—	—	Several cases under	personal observation, referred	to	
Taylor and Collier	BRAIN, Winter No., 1901	F.	56	Myxoma	C. 3		+
		M.	34	Tumour	C. 5		
		M.	48	? Tumour	C. 5		
		F.	14	Spinal caries	Cervical		
		M.	28	Fracture Dislocation	C. 6		
		M.	24	Myelitis	Cervical		
		M.	36	,,	Upper dorsal		
		M.	34	,,	D. 2		
		F.	22	,,	D. 2		
		F.	15	? Hæmatomyelia	C. 4		
		M.	41	Chronic Lesion ?	C. 5		
		M.	28	Myelitis	Upper dorsal		
Thorowgood	*Clin. Trans.*, viii., 1875, p. 80	—	—	Trauma to neck	Cervical		
Uhthoff	*Berl. Klin. Woch.*, 1885, No. 16	—	—	Three cases of "multiple sclerosis" with optic neuritis			

A CONTRIBUTION TO THE CLINICAL SIGNIFICANCE OF ABSENCE OF THE TENDO-ACHILLIS-JERK.

BY EDWIN BRAMWELL, M.B., M.R.C.P.LOND. AND EDIN.

CONTENTS.

(1) Introductory remarks.
(2) Method of examining the Achillis-jerk.
(3) The Achillis-jerk in health and disease; an investigation based upon the examination of 1,009 individuals.
(4) Conclusions.

INTRODUCTORY REMARKS.

THE following investigation was undertaken in the hope of ascertaining more exact knowledge as to the diagnostic significance of absence of the Achillis-jerk.

It is only within the last two or three years that any diagnostic value has been attached to this jerk, and that probably for two reasons. Firstly, since in the majority of cases in which the Achillis-jerk is altered, the knee-jerk is similarly affected, the examination of the Achillis-jerk consequently affords little or no additional diagnostic data; while, secondly, the idea is commonly prevalent that the Achillis-jerk is absent or not obtainable in a certain proportion of healthy individuals.

The first question which it is proposed to consider in this paper is the state of the Achillis-jerk in "health"; in other words, is absence of the Achillis-jerk always to be regarded as pathological? So long as doubt exists upon this point, it is obvious that absence of the Achillis-jerk will never be regarded as a physical sign of diagnostic importance.

In the latter part of the paper, the results of the examination of a number of patients suffering from various forms of disease are recorded, and conclusions drawn as to the diagnostic value of absence of the jerk.

Before referring to the state of the Achillis-jerk in health and disease, it is, however, advisable to describe the methods best adapted for examining the jerk. This is all the more necessary since it appears probable that defective technique in examining the jerk is largely accountable for the little attention which it has hitherto received, as well as for the uncertainty which exists regarding its clinical significance.

METHOD OF EXAMINING THE TENDO-ACHILLIS-JERK.

The kneeling position, as recommended by Babinski, is without doubt that in which the Achillis-jerk is most conveniently examined and easily obtained.

The patient kneels on a chair with his feet projecting a few inches over the edge. In this position the muscles of both the front and back of the leg are usually completely relaxed. A hammer of some kind is necessary with which to strike the tendon. A wooden stethoscope with a rubber ring attached around the ear-piece serves very well for this purpose. If no hammer is at hand the edge of a thin book may be used.

The tendon is struck *sharply*, close to its insertion into the os calcis, and, if the jerk is present, the stroke is immediately followed by a quick plantar flexion-movement of the foot at the ankle joint.

The true jerk is a quick movement occurring immediately *after* the stroke, and is quite distinct from the slight movement of the foot which occurs as a mechanical effect in cases in which the reflex is lost.

If the jerk is not obtained, the observer must make sure that the muscles of the legs are not contracted. It is especially important in comparing the jerks on opposite sides of the body, to see that the leg muscles on both sides are fully and equally relaxed.

The various methods of reinforcement recommended by Jendrassik,[1] such as pulling on the hands, &c., should be adopted where the jerk is not obtained. A succession of sharp strokes sometimes elicits the reflex when a single stroke fails to do so. In one case in which the Achillis-jerk was difficult to get, Dr. Byrom Bramwell found that striking the calf muscle several times with the hammer appeared to reinforce the jerk. The Achillis-jerk, when present, can usually be readily obtained with boots on, in the kneeling position.

When the patient is unable to kneel, the examination must be made in the prone position.

If the jerk is well marked, it is easily obtained in this position. This method of examination is, however, much more difficult than the kneeling position, and its successful application requires considerable experience. The following is the procedure which the writer adopts, when it is necessary to examine the patient in the prone position.

The patient is asked to turn from his back slightly towards the side of the limb about to be examined. The limb is flexed at the knee almost to a right angle and is then laid on its outer side upon the bed. In this way, complete muscular relaxation is usually secured. The ball of the great toe is grasped lightly between the finger and thumb of the left hand and the foot is dorsi-flexed sufficiently to put the tendo Achillis slightly on the stretch. The inner edge of the tendon is then struck with the hammer, and the slightest plantar movement of the foot is felt by the hand which grasps the patient's foot. Should no jerk be obtained, the tension on the tendon should be slightly increased and the jerk again tested. Reinforcement should then, if necessary, be employed.

THE STATE OF THE TENDO-ACHILLIS-JERK IN HEALTH AND DISEASE.

The following investigation is based upon an examination of 1,009 persons from various communities:—

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[1] *Neurolog. Centralblatt*, 1885, S. 412.

Fifty Children from the Medical Wards of a Children's Hospital.

Through the kindness of Drs. Burn Murdoch, Melville Dunlop and John Thomson the writer had the opportunity of examining the Achillis-jerks in 50 patients in the Royal Hospital for Sick Children, Edinburgh; none of them, so far as is known, were suffering from any form of nervous disease.

Almost all of these cases were examined in bed in the prone position, and in every case, with one exception, both Achillis-jerks were at once obtained. The inability to obtain the jerks in the single exceptional case was due to the fact that the child, who had recently been admitted, was fretful and would not relax its leg muscles. On a subsequent occasion, both jerks were found to be quite active in this case.

Two Hundred and Nineteen School Children, Eight to Fifteen Years of Age, including 111 *Deaf-mutes.*

Dr. Lockhart Gillespie and the House-Governor of Donaldson's Hospital, Edinburgh, kindly gave the writer permission to examine the inmates of that Institution.

The Achillis-jerks were examined in 219 children, varying in age from eight to fifteen years. Of these children, 111 (60 boys and 51 girls) were deaf-mutes, while the remaining 108 (67 boys and 41 girls) were healthy school children. All were examined in the kneeling position.

Both Achillis-jerks were readily obtained in every case with four exceptions; all of whom were boys and all deaf-mutes. In none of these four cases in which the Achillis-jerks were both absent was there rigidity of the leg muscles. A day or two later, the four exceptional cases were again examined, and in one the jerks were obtained, though with difficulty. In the remaining three the absence of the Achillis-jerks was confirmed. In one of these cases, the knee-jerks were absent, in another they were very sluggish, while in a third they were quite active. In none of these cases was any cause discovered to account for the absent Achillis-jerks.

One Hundred Convalescent Fever Cases.

The writer's thanks are due to Dr. Claude B. Ker, who kindly gave him the opportunity of examining 100 convalescent patients in the City Fever Hospital, Edinburgh, the great majority being children and young adults.

The patients were convalescing from the following fevers:—scarlet fever (69 cases), enteric (14), diphtheria (10), erysipelas (5), and quinsy (2).

In all the cases, with four exceptions, both the knee and Achillis-jerks were present. In the case of two patients who were suffering from post-diphtheritic paralysis, both the knee and Achillis-jerks were absent. In one instance (a convalescent from scarlet fever), the left Achillis-jerk was not obtained, but two days later was found to be well marked. In the last case, also post-scarlatinal, neither Achillis-jerk could be obtained although the knee-jerks were brisk. The inability to obtain the jerks in this case was ascribed at the time to rigidity of the leg muscles, but, unfortunately, the writer had not a further opportunity of examining the case as the patient left the hospital on the following day.

Eighty Medical Students.

Eighty medical students were examined in the kneeling position with boots or shoes on; in every case but one both jerks were well marked. In this instance, they were obtained after the student had removed his boots; reinforcement, however, had to be resorted to before one of the jerks became evident. The knee-jerks were somewhat sluggish in this case.

Two Hundred and Thirty Individuals Over 40 *Years of Age.*

Through the kindness of Dr. Edward Carmichael and Dr. R. S. Aitchison, and the Governors of the Craiglockhart and Craigleith Poorhouses, Edinburgh, opportunity was afforded of examining 230 inmates of these institutions, including 157 men and 73 women.

The great majority of the individuals examined were well past middle age. *The jerk was tested in all cases in the kneeling position, the patients wearing either light shoes or stockings only.* Reinforcement was used in all cases where the jerks were difficult to obtain. The results in relation to age are interesting and are well shown in the following table :—

The Achillis-jerk in Persons over 40 *Years of Age.*

ANALYSIS OF 230 CASES.

Age	No. of Cases.	Cases in which Achillis-jerks not obtained.			Percentage of Cases in which Achillis-jerks affected at different ages.
		One.	Both.	Total.	
Over 80 years	15	2	10	12	80 per cent.
70-80 years	65	5	36	41	63 ,,
60-70 ,, ...	80	3	25	28	35 ,,
50-60 ,, ...	42	1	4	5	12 ,,
40-50 ,, ...	28	—	—	—	—

It is probable that if these cases had been again examined, in some the Achillis-jerks would have been obtained; the percentages in the last column are for this reason almost certainly too high. Admitting this, however, a justifiable conclusion to be deduced from this investigation is, that in persons past middle age, the Achillis-jerk is not always obtainable, and further, with increasing years the proportion of cases in which the Achillis-jerk is absent becomes increasingly greater.

In old people, the Achillis-jerk when obtained is usually much less active than in young or middle aged adults. In old people in whom the Achillis-jerks are well marked, the individuals are often unusually active and frequently appear to be endowed with a vitality beyond their years.

One Hundred Insane Patients.

Through the kindness of Dr. Clouston, the writer had the opportunity of examining the Achillis-jerks in 100 male patients in the Royal Asylum, Morningside, Edinburgh. In these cases, both Achillis-jerks were present in eighty-nine cases, while one or both were absent in eleven cases. Of the eleven cases in which the Achillis-jerks were absent, five were cases of general paralysis, five were cases of senile dementia, and one was a case of alcoholic insanity.

In three of the cases of general paralysis, both the Achillis-jerks and knee-jerks were absent. In one case, the right, in another the left, Achillis-jerk was absent, the knee-jerks and opposite Achillis-jerk being present. In one case, the right knee-jerk was absent, the left knee-jerk and both Achillis-jerks being present.

In all of the five cases of senile dementia included in the eleven exceptional cases, both knee-jerks were present; while in four, both Achillis-jerks were absent. In the remaining case, the left Achillis-jerk was present. All of these patients were well advanced in years, four being from 63 to 66 years of age. The fifth patient stated that he was 55, but his appearance was that of a very much older man.

In the case of alcoholic insanity both Achillis-jerks were lost, the knee-jerks being unaffected.

One Hundred Patients from the Medical Wards of a General Hospital.

For kind permission to examine 100 hospital patients in the Edinburgh Royal Infirmary, suffering from a variety of medical diseases, the writer's acknowledgments are due to Professor Wyllie, Drs. Byrom Bramwell, Alexander Bruce and Alexander James.

The patients examined were suffering from the following conditions :—

	No of cases.	One or both Achillis-jerks absent or greatly diminished.
Cardiac disease of various forms	16	1
Rheumatism	14	1
Dyspepsia, including gastric ulcer	13	2
Pneumonia	11	0
Diabetes Mellitus	7	2
Phthisis	7	0
Chlorosis	6	0
Aortic aneurism..	4	2
Acute Bright's disease	3	0
Exophthalmic goitre	3	0
Pernicious anæmia	2	0
Diabetes insipidus	2	0
Cancer of liver	2	0
Pericarditis, psoriasis, lupus, leucocythæmia, cancer of stomach, arrested bodily development, chronic bronchitis, gout,* catarrhal jaundice. and nocturnal eneuresis—of each 1 case	10	1*
Total	100	9

Both Achillis-jerks were present and well marked in all but nine cases.

The following is a brief record of the exceptional cases :—

Case 1.—J. J., labourer, aged 37 (under the care of Dr. B. B.), suffering from diabetes mellitus. Both Achillis- and knee-jerks were absent.

Case 2.—J. B., iron-planer, aged 34 (under the care of Dr. B. B.), suffering from diabetes mellitus. Both Achillis-jerks and knee-jerks were absent.

It is a well known fact that the deep reflexes are often absent in diabetes mellitus. In one case examined by the writer, the knee-jerk was absent and the Achillis-jerk present (the patient had only one leg). Babinski[1] has examined a diabetic in whom the knee-jerks were normal, while the Achillis-jerks were lost.

Case 3.—R. S., joiner, aged 34½ (under the care of Dr. B. B.), complained of dyspepsia from which he had suffered for seventeen years, he had been worse since a severe attack of influenza ten months previously. Both the knee- and Achillis-jerks were absent.

He was a temperate man as regards alcohol, and absolutely denied syphilis and gonorrhœa; there was no history of secondary syphilitic symptoms. He had been married ten years; his wife had had a miscarriage at six weeks, a few months after marriage, which was said to have been caused by a fall. There was one child alive which was said to be perfectly healthy: there had been two miscarriages at a later date, attributed by a gynæcologist to some uterine trouble. There were no signs of tabes or G. P. I. The pupils were equal, regular and reacted well to light. There was no ataxia or Rombergism and no thoracic or ulnar analgesia. No history of shooting pains or bladder trouble. His mental state appeared perfectly satisfactory. There was no weakness of the legs or tenderness of the calves and no history of diphtheria. No sugar in the urine.

Possibly the loss of the knee- and Achillis-jerks may have been the result of the attack of influenza above referred to (?).

Case 4.—J. C., labourer, aged 48, was admitted into the R. I. E. (under the care of Dr. B. B.), suffering from a typical gouty swelling of the left hand which rapidly disappeared under appropriate anti-gouty remedies.

In this case both Achillis-jerks were absent while the knee-jerks were present although not well marked. There was no history of syphilis.

On enquiry, it was ascertained that *the patient had had two separate attacks of sciatica* between two and three years previous to his admission to hospital, affecting first one leg and then the other.

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[1] *Revue Neurologique*, 1901, p. 482.

The occurrence of absence of the Achillis-jerk in cases of sciatica will be referred to later (see p. 570).

Case 5.—H. F., labourer, single, aged 38 (under the care of Dr. B. B.), was admitted to the R. I. E. on account of dyspeptic symptoms.

The patient complained of shooting pains in both legs below the knee.

On examination it was found that the knee-jerks were active and equal but that the Achillis-jerks were both absent.

The calf muscles were exceedingly tender, but there was no perceptible weakness of the extensors of the feet. The patient stated that he had been drinking heavily for some months previously, and he himself attributed his symptoms (dyspepsia, &c.), to this cause.

The patient admitted having had gonorrhœa, but denied syphilis, and there was no history of secondary symptoms. A thorough examination failed to elicit any signs suggesting tabes—the pupils reacted actively to light, there was no thoracic or ulnar analgesia, no affection of the bladder or rectum, and no derangement of the sexual functions.

A diagnosis of *early peripheral (alcoholic) neuritis* seemed perfectly justifiable on these facts.

The condition of the Achillis-jerk in peripheral neuritis is referred to on p. 569.

Case 6.—A. F., domestic servant, aged 25. This patient was recently admitted to the R. I. E. (under the care of Dr. B. B.) supposed to be suffering from subacute rheumatism. She complained of pains in her legs and back. The history of her illness was as follows:—Six weeks previously she first noticed a sharp pain behind the right knee which afterwards extended up and down the leg, and in the course of a few days, spread to the opposite leg. At the same time she had a severe pain in the lower part of the back. As the pains did not disappear with anti-rheumatic remedies, she was recommended for admission to the hospital. There had been no swelling nor pain in any of her joints. She had not been feverish, nor had she sweated. She had never suffered from rheumatism nor was there any history of injury to the back. There was no family history of rheumatism or tubercle. On examination she was a well nourished, healthy-looking girl. Her temperature was normal, her tongue clean and skin dry. There was no swelling of any of the joints. The pain which she complained of was situated over the upper sacral region and was, she said, made worse by movement. The pains in the legs, which were not so severe as they had been, affected both legs equally, and were felt down the back of the legs, especially behind the knees. The pains were shooting in character. There was slight tenderness on pressure over the sciatic nerves. The power of the muscles of both the front and back of the leg was markedly diminished, as was that of the hamstrings on both sides, while no distinct weakness could be made out in the extensors of the knee, the muscles of the abdomen, or upper limbs.

A very careful examination failed to reveal the slightest evidence of impairment or hyperacuity of sensation either to touch or pain over the lumbar or sacral root areas. There was no tenderness of the leg muscles. The knee-jerks were somewhat increased as was the left Achillis-jerk, while the *right Achillis-jerk was absent.* There was a double extensor response, best marked on the left side. There had been no disturbance of the bladder reflex, no delay in commencing the act, no incontinence, no precipitate micturition. Some weeks previous to admission, she had for a few days experienced a burning sensation when passing water The patient stated that she had no vaginal discharge. The rectal functions were unaffected. There was no irregularity of the spine and no tenderness on pressure. The functions of the special senses, cranial nerves and upper extremities were perfectly normal.

In this case the weakness of the hamstring and leg muscles, together with the pains in the back and legs, and the double extensor response, pointed to a *local lesion involving the last lumbar (?) and upper sacral roots,* or that part of the spinal cord with which they are directly connected. What the exact nature of this lesion was it was difficult to say. *In this case the absent Achillis-jerk on the right side was of undoubted localising value.*

Case 7.—J. D., mason, aged 53 (under the care of Dr. B. B.), admitted to the R. I. E. with definite signs of a thoracic aneurism. He had never been a heavy drinker.

The right Achillis-jerk was present though difficult to elicit, the left Achillis-jerk on the first occasion on which it was tested could not be obtained, but subsequently, when the patient was examined in the kneeling position, a very slight movement of the foot was once or twice seen.

The knee-jerks were increased and equal.

There was a double extensor response.

The right pupil was slightly larger than the left and oval in shape, the result of an old iritis. *Both pupils reacted very slightly to light, rather better on accommodation.*

The patient admitted having had gonorrhœa thirty years previously, but denied syphilis, and there was no history of secondary symptoms. No history of sciatica or shooting pains in the legs; no thoracic or ulnar analgesia. Some dribbling of urine lately, but this may have been accounted for by an enlarged prostate.

The patient died soon after leaving the hospital. At the autopsy two large aneurisms were found, one of them, situated opposite the bodies of the ninth, tenth and eleventh vertebræ, had eroded these considerably. The arteries were extremely atheromatous.

Case 8.—A., aged 57 (under the care of Professor Wyllie), suffering from aortic incompetence.

Both Achillis-jerks were absent (examined on two occasions, both in the kneeling and prone positions), while the knee-jerks were present and about normal in activity.

The patient denied syphilis, and there was no clear history of secondary symptoms. *His pupils, however, were slightly irregular and scarcely reacted at all to light, though well on accommodation.* There were no other signs suggestive of tabes or G. P. I.; no evidence of peripheral neuritis, and no history of sciatica. His wife had had thirteen children, *six of whom died in infancy.*

Case 9.—J. R., labourer, aged 34 (under the care of Dr. B. B), suffering from a thoracic aneurism. Both Achillis-jerks were absent. The activity of the knee-jerks was below the normal, the right being less active than the left. The pupils measured 2½ mm., were equal and regular and reacted to light, although the contractions were not very brisk. They contracted actively on accommodation. There were no other signs suggesting tabes and nothing pointing to peripheral neuritis. He had not had sciatica. The patient was married, but had had no children. He denied syphilis. Unfortunately, he left the hospital before additional data bearing upon the question of previous syphilis had been enquired into.

Although in none of the three cases last described is there absolutely certain evidence of previous syphilis, the writer suggests syphilis as the probable cause of the loss of the Achillis-jerks in these cases.[1] In other words, he believes that loss of the Achillis-jerk may occur as a result of syphilis unaccompanied by other signs of tabetic degeneration, just as the Argyll Robertson pupil is sometimes observed as a sequel of syphilis in the absence of other signs of tabes, G. P. I., &c.

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[1] In connection with these cases it is interesting to note that Charles L Greene, of St. Paul, in his work on "Examination for Life Insurance," referring to the knee-jerk, remarks: "The author has several times found it to be lacking in aneurism of the aorta." Rebman Limited, London, 1901.

The data upon which the writer bases this statement are as follows:—

(1) It is very exceptional to meet with absence of the Achillis-jerk, where a satisfactory explanation cannot be ascertained to account for it.

(2) The three cases mentioned above were all suffering from aortic disease (two from aortic aneurism), a fact which suggests some common etiological factor.

(3) Syphilis is an important factor in the production of aneurism and aortic disease.

(4) Although in none of the three cases above mentioned is there absolute evidence of syphilis, the aneurismal or aortic lesion in itself makes it not improbable that the patients had at some previous time suffered from syphilis. Further, in two of the three cases, apart from the aortic disease, there are other facts which point towards the probability of syphilis, notably the condition of the pupils, and in one of the cases the large number of deaths in infancy.

(5) In a case which will be referred to later (p. 568), the only explanation which could be found to account for the absence of the jerks was previous syphilis, and in this case there was a definite history of the disease.

(6) It has been pointed out by Babinski[1] that the Achillis-jerk is often lost before the knee-jerk in tabes.

ONE HUNDRED CASES OF NERVOUS DISEASES.

The Achillis-jerks were examined in 100 patients suffering from various forms of nervous disease under the care of Dr. Byrom Bramwell. The following table specifies the particular diseases in question, while a separate column shows the number of cases in which either one or both

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[1] *Revue Neurologique*, 1901, p. 482.

Achillis-jerks were absent, or greatly diminished in activity:—

	No. of Cases.	Achillis-jerks lost or greatly diminished.
Disseminated sclerosis	13	0
Hysteria	11	0
Intracranial tumour	10	0
Peripheral neuritis	9	6
Chorea	9	0
Epilepsy	8	0
General paralysis of the insane	4	1
Hemiplegia	4	0
Sciatica	4	2
Spastic paraplegia	4	0
Neurasthenia	3	0
Spinal caries	3	0
Jacksonian epilepsy, auditory vertigo, local neuritis of upper limb, infantile paralysis,* syringomyelia, and Bell's palsy*—2 cases of each	12	2*
Pseudo-hypertrophic paralysis,† tubercular meningitis, miner's nystagmus, cerebral syphilis, lesion of cauda equina,† and a case of defective mental development —1 case of each	6	2†
Total	100	13

It will be seen from this table that the Achillis-jerks were present and marked in all but thirteen cases. Either one or both Achillis-jerks were absent or greatly diminished in six cases of peripheral neuritis, in two cases of sciatica and in one case of each of the following conditions, viz., pseudo-hypertrophic paralysis, infantile paralysis, a lesion of the cauda equina, general paralysis of the insane and Bell's palsy. It will be convenient to refer firstly to these single exceptional cases, considering afterwards the state of the Achillis-jerks in sciatica and in peripheral neuritis.

Case 1.—A. H., schoolboy, aged 11, suffering from *pseudo-hypertrophic paralysis.* Both the knee-jerks and Achillis-jerks were present, although greatly diminished in activity and somewhat difficult to obtain. The state of the jerks in this case was only what was to be expected from the *weak condition of the leg muscles.*

Case 2.—J. G., van-driver, aged 24, suffering from the results of an attack of *acute anterior poliomyelitis.* The right Achillis-jerk was absent, the left present, the right knee-jerk being active, while the left was absent. *The wasted condition of the right calf* and left thigh muscles was obviously responsible for the absent jerks in this case.

Case 3.—D. H., labourer, aged 48, was admitted to hospital suffering from a *lesion of the cauda equina.* His illness had commenced six years previously with pains in the legs, and the condition had progressed for twelve or fifteen months, since which time his symptoms had remained *in statu quo.* A definite history of syphilitic infection followed by characteristic secondary symptoms was obtained. The history and distribution of his symptoms at the time of examination pointed to a lesion involving the sacral roots. Neither Achillis-jerk was obtained, the left knee-jerk was difficult to get, the right was absent. In this case, *the great wasting of the calf muscles* afforded an obvious explanation to account for the lost jerks.

Case 4.—J. R., an old soldier, aged 34, with a typical Bell's palsy of ten days' duration. On testing the Achillis-jerks, it was found that only the slightest movement of either foot was obtained even with reinforcement. The knee-jerks were present but feeble. The pupils reacted well to light and a thorough examination failed to reveal any signs suggestive of tabes, general paralysis or peripheral neuritis. There was no history of sciatica. The patient gave a definite history of hard sore some ten years previously, for which "black wash" had been ordered; there appeared to have been no secondary symptoms. *Syphilis* was probably the cause of the defective Achillis-jerk in this case.

Case 5.—G. K., aged 32, admitted to the R. I. E. suffering from *general paralysis of the insane.* His symptoms, which were sufficiently typical to permit of a positive diagnosis, need not detain us. The knee-jerks were abnormally active, the left Achillis-jerk was absent and the right distinctly diminished. This patient had had *syphilis* followed by secondary symptoms.

The Achillis-Jerks in Peripheral Neuritis.

It will be seen from the table on page 567 that nine cases of peripheral neuritis were examined. Of these cases seven were cases of alcoholic neuritis.

In four cases of alcoholic neuritis (one man and three women), both the knee and Achillis-jerks were absent.

In the three remaining cases the knee and Achillis-jerks were present; in one case the knee-jerks were exaggerated. These three patients were all males, all had been drinking heavily, and in all there were pains in the legs and marked tenderness of the calf muscles. With regard to the classification of these cases, the objection might be raised that the evidence upon which the diagnosis of neuritis was based is not sufficiently strong. Even if it be granted that this is so, their inclusion seems justifiable in order to show that pains in the legs and marked hyperalgesia of the calves may exist as a result of alcoholism in association with normal Achillis-jerks.

In the case referred to on page 563 the condition would appear to have advanced a stage further, for in that case the Achillis-jerks were both absent.

Although from a single case such as that just mentioned one would hesitate to make the general statement that the Achillis-jerks are lost, or sometimes lost, before the knee-jerk in alcoholic neuritis, since there is always the possibility of some additional unrecognised etiological factor having been present, still the loss of the Achillis-jerks in this case *suggests* that such may be the case.

Two cases classed as peripheral neuritis still remain for consideration.

The first case is that of a sailor, aged 41, who was shown to the Medico-Chirurgical Society of Edinburgh by Dr. Byrom Bramwell.[1] The patient was suffering from a peculiar form of muscular atrophy of *peripheral distribution* commencing in the lower limbs and afterwards affecting the upper. The onset was gradual and associated with cramps in the muscles. There was no objective disturbance of sensation and no affection of the bladder. The knee-jerks were feeble and the Achillis-jerks absent on both sides. *The absence of the Achillis-jerks was obviously due to the great wasting of the calf muscles* (all power of movement was completely lost in all the muscles below the knee on both sides). The diagnosis in this case was somewhat uncertain. Beri-beri was considered as a possibility, but finally excluded. In some respects the case resembled the peroneal type of muscular atrophy. A very similar case has been described by Mills.[1]

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[1] *Trans. Med. Chirurg. Soc. of Edinburgh*, 1901, p. 146.

In a second case, which was classed as peripheral neuritis, both the Achillis-jerks were absent, while the knee-jerks were present though sluggish. This case presented many peculiar features, and the etiology of the condition was quite obscure. Space does not, however, permit of a detailed description of it.

THE ACHILLIS-JERKS IN SCIATICA.

The Achillis-jerks were examined in four cases of sciatica; in two of these cases they were affected and in two normal. The clinical features of the two cases in which the jerks were affected were briefly as follows:—

Case 1.—A labourer, aged 30, was admitted to hospital suffering from left-sided sciatica; the left Achillis-jerk was absent, the right although present was distinctly diminished; the knee-jerks were equal and of about normal activity. Upon enquiry, it was ascertained that, three years previously, he had had a severe attack of sciatica on the *right* side.

Case 2.—A farmer, aged 36, was admitted to hospital suffering from left-sided sciatica; the left Achillis-jerk was absent, while the right was difficult to obtain. Both knee-jerks were about normal, the left perhaps a shade less marked than the right. This patient had not previously suffered from sciatica.

It was pointed out by Babinski, in 1896, that in cases of sciatica the Achillis-jerk is usually abolished on the affected side. This observation has been confirmed by other observers. According to Max Biro[1] this alteration of the Achillis-jerk —abolition or diminution—which occurs in sciatica, is met with only on the affected, never on the sound side. The presence of the alteration of the reflex in a case of sciatica, indicates that the condition is in reality a neuritis of the sciatic nerve.

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[1] *Journal of Nervous and Mental Disease*, 1900, p. 324.

Max Biro[2] states that in cases of sciatica the lost reflex returns gradually with recovery. It appears, however, from one of the cases described above that years may elapse before the normal activity of the jerk is regained.

In the case of J. C., referred to on page 562, in which the loss of both Achillis-jerks could only be accounted for by two separate attacks of sciatica, the Achillis-jerks were both absent when the patient came under observation two years later.

The absence of the Achillis-jerk must be regarded as a valuable sign in distinguishing true sciatica from the hysterical variety.

THE ACHILLIS-JERKS IN TABES DORSALIS.

The writer has examined the Achillis-jerks in thirty cases of tabes seen in the hospital practice of his father.

In twenty-five of these cases both the knee-jerks and Achillis-jerks were absent.

The condition of the jerks in the five exceptional cases was as follows:—

(1) Both knee- and Achillis-jerks increased.

(2) Both knee- and Achillis-jerks present and about normal.

(3) Both knee-jerks and one Achillis-jerk active, the other Achillis-jerk constantly diminished.

(4) Both knee-jerks active, one Achillis-jerk absent, the other slight.

(5) Both knee-jerks and one Achillis-jerk absent, the other Achillis-jerk being active.

Thus of four tabetics in whom the knee-jerks showed no

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[1] *Deutsche Zeitschrift f. Nervenheilkunde*, 1901, S. 188. [2] *Loc. cit.*

diminution, in two the absence or constant diminution of the Achillis-jerk afforded diagnostic data; in one of these cases the information derived being of undoubted value in substantiating the diagnosis. The following is a brief record of this case:—

J. C., aged 40, a sailor, complaining of double vision which had lasted for six months. The patient had had *syphilis* twelve years previously, followed by secondary symptoms. He had not had sciatica.

For three or four months he had had *occasional slight shooting pains in the legs.* There had been some *delay in micturition,* and he stated that occasionally the urine dribbled away at night. The water comes in a good stream. He had had some *diminution of sexual desire and power* during the last few months.

On examination, it was found that there was *complete paralysis of the right third nerve and that neither pupil reacted* to light. There was no ataxy and no Rombergism. No analgesia was detected in the arms, legs or trunk. The knee-jerks were brisk and equal. *The right Achillis-jerk was marked, the left distinctly sluggish.* There was a double flexor response. His memory was good and no mental peculiarity was noticed. The optic discs were well coloured and their edges sharp. There was no tremor of the tongue or hands, no twitching about the face and no affection of speech.

In the great majority of tabetics both the knee- and Achillis-jerks are lost at the time they first come to the physician.

Goldflam,[1] writing upon tabes in 1888, mentions incidentally the Achillis-jerk, which, he says, is generally abolished at the same time as the knee-jerk.

Ostankow[2] has recorded twenty-six cases in which he examined both the knee- and Achillis-jerks; in all of these cases both jerks were absent.

Babinski,[3] in the same year, stated to the Soc. Méd. des Hôp. de Paris, that in tabes the Achillis-jerk is sometimes lost before the knee-jerk, and that the absence of the Achillis-jerk will help the diagnosis in such a case. He had met with two cases in which the knee-jerks were lost while the

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[1] Goldflam, *Neurolog Centralblatt*, 1888, S. 528, u. 556.
[2] Ostankow, *Neurolog Centralblatt*, 1898, S. 140.
[3] *La Semaine Médicale*, 1898, p. 489.

Achillis-jerks were still present; on the other hand, he had seen three cases where the Achillis-jerks were absent, and the knee-jerks present.

Mills,[1] in a paper read before the New York Neurological Society, January 3, 1899, refers to a case of tabes in which the absence of both Achillis-jerks "was of decided value in reaching a correct diagnosis." "This was a case sent to me for opinion by two physicians. The diagnosis of neurasthenia had been given, and the existence of tabes was regarded as in doubt, chiefly for the reason that both knee-jerks were present, although the patient had a recent history of what appeared to be shooting pains, and some evidences of probable slight involvement of the bulbar nuclei. Ataxia was not present, but the tendo Achillis-jerk was lost on both sides, quadriceps-jerk, knee-jerk, and gastrocnemius-jerk being present. I concluded that the case was organic, probably a somewhat irregular form of tabes."

Babinski[2] states that since his attention has been especially directed to the question of the Achillis-jerk in tabes, he has observed five cases of affection of the knee-jerk with a normal Achillis-jerk in that disease, whereas he has seen forty patients (*une quarantaine de malades*), undoubted tabetics, in whom the Achillis-jerk was alone affected, or was affected to a greater degree than the knee-jerk. He has come to the conclusion that in the first stages of the disease the Achillis-jerk affords data for diagnosis more frequently than the knee-jerk.

Although the literature upon the subject is as yet small, the above observations will serve to show that *the Achillis-jerk should always be examined in a suspected case of early tabes in which the knee-jerks are still present, and that in such cases its absence may be of considerable value in making the diagnosis.*

The loss of the Achillis-jerk in general paralysis is, no doubt, to be accounted for by an associated "tabetic" lesion; it may prove of value in the diagnosis of early cases of this disease.

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[1] *Journal of Nervous and Mental Disease*, 1899, p. 159.

[2] *Revue Neurologique*, 1901, p. 483.

Table Illustrating the State of the Achillis-jerk in 1,009 *Cases of Health and Disease.*

	Class of Case.	No.	One or both Achillis-jerks absent or greatly diminished.	Remarks regarding cases where Achillis-jerks were absent or diminished.	Cause of loss or diminution of Achillis-jerks.
1	Sick children	50	0		
2	School ,,	219	4	All boys and all deaf-mutes	?
3	Fever convalescents	100	3	2 Post-diphtheritic paralysis	Post-diphtheritic neuritis
				1 Convalescence from scarlet fever	Leg muscles not properly relaxed
4	Medical students	80	0		
5	Insane ..	100	11	5 General paralysis ..	G.P.I.
				5 Senile dementia ..	Senility
				1 Alcoholic insanity ..	Previous alcoholic neuritis
6	Persons over 40 years of age from Workhouse Population	230	86	All over 50 years of age. Frequency of absence increases with increasing years (see Table, p. 559)	Senility
7	Hospital Cases (General Medical)	100	8	2 Diabetes mellitus ..	Diabetes mellitus
				1 Dyspepsia	Early alcoholic neuritis
				1 Dyspepsia	Influenza?
				1 Gout	Previous sciatica
				2 Aortic aneurism 1 Aortic incompetence	Syphilis?
8	Hospital Cases (Nervous, exclusive of tabes)	100	13	6 Peripheral neuritis	Peripheral neuritis
				2 Sciatica	Sciatic neuritis
				1 Pol. ant. acuta 1 Pseudo-hypertrophic paralysis 1 Lesion of cauda equina	Atrophy of calf muscles
				1 Bell's palsy ..	Syphilis
				1 G.P.I.	
9	Hospital Cases (Tabes dorsalis)	30	28	25 Both knee-jerks and Achillis-jerks absent 1 Knee-jerks active; right Achillis-jerk absent; left Achillis-jerk slight 1 Knee-jerks active; right Achillis-jerk active; left Achillis-jerk constantly diminished 1 Knee-jerks absent; right Achillis-jerk active; left Achillis-jerk absent	Tabetic degeneration of afferent neurons
	Total	1009	cases		

CONCLUSIONS.

(1) The Achillis-jerk is most easily and conveniently obtained in the kneeling position. The examination in the prone position is more difficult and requires considerable experience, although the results obtained in this position are almost equally reliable in the hands of a skilled observer.

(2) The Achillis-jerk is (probably) constantly present in "health" in persons under 50 years of age. In other words the loss of the Achillis-jerk in a person under 50 years of age (in the absence of any local cause, such as great œdema of the legs, which may mechanically interfere with the examination) is always to be looked upon as *a sign of organic disease,* with the same assurance as loss of the knee-jerk is so regarded at the present time.

(3) In persons over 50 years of age the activity of the Achillis-jerk diminishes with increasing age (see table, p. 559), consequently in old people absence of the Achillis-jerk, especially if bilateral, cannot be regarded as having the same diagnostic significance that it has in children, young persons and middle-aged adults.

(4) In the majority of cases of disease in which the Achillis-jerks are absent, the knee-jerks are also lost, *e.g.*, most cases of tabes, peripheral neuritis, &c.

(5) In certain conditions in which the knee-jerks are unaltered the Achillis-jerks are found to be absent or greatly diminished. Under these circumstances loss of the Achillis-jerk may prove a valuable adjunct to diagnosis.

(6) Since in tabes the Achillis-jerk is usually lost before the knee-jerk, its condition should always be ascertained in suspected cases of that disease (Babinski), and more particularly in those cases in which the knee-jerks are present.

(7) It has been shown by Babinski and others that in cases of sciatica the Achillis-jerk is usually lost on the affected side; the loss of the Achillis-jerk proves the existence of a sciatic neuritis. The absence of the Achillis-jerk is for this reason a physical sign of importance in the diagnosis between true sciatica and the hysterical form of the disease.

(8) The loss of the Achillis-jerk caused by a sciatic neuritis may persist for years after the sciatica has itself disappeared.

(9) Cases of multiple neuritis are met with in which the Achillis-jerks are absent, while the activity of the knee-jerks remains unimpaired. In such cases the absence of the Achillis-jerks is of value in diagnosis.

(10) It appears probable that the Achillis-jerk is sometimes lost as a result of syphilis, just as the Argyll Robertson pupil sometimes occurs as a result of that disease unaccompanied by other signs of tabes or general paralysis.

(11) The absence of the Achillis-jerk is sometimes of value in localising lesions in the spinal cord or the nerve roots.

ON THE SEAT OF THE PSYCHO-PHYSICAL PROCESSES.

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THE term "psycho-physical process" has been used with various meanings by different authors; it is important therefore to define at once the sense in which it is used in the title of this paper and throughout the following pages. By psycho-physical process I mean to denote a physiological process that is immediately correlated with and invariably accompanied by a psychical process. In this usage I follow Prof. G. E. Müller,[1] the distinguished psychologist of Gottingen, on whom, as regards all things psycho-physical, the mantle of G. T. Fechner may be said to have fallen. I would prefer to define it more fully as that part of the total process of physiological excitation within the nervous system which stands in a relation of immediate interaction with psychical process or consciousness. But this latter definition, involving as it does the rejection of the hypothesis of psycho-physical parallelism, would be unacceptable to the very numerous upholders of that doctrine, and the former is sufficient for the purpose of this essay. Now, whether we hold by the doctrine of psycho-physical parallelism or by the hypothesis of interaction between physical and psychical processes, we have to recognise that not all of the processes that constitute the activity of the brain at any moment of consciousness are psycho-physical processes in the sense defined above; it is, for example, generally admitted that the medullated fibres of the white matter of the brain and cord are entirely similar in nature and function to the fibres of the peripheral nerves, that their function is the transmission, and possibly the augmentation, of the state of excitation, and that this process is not immediately correlated with psychical process.[1] It becomes then a matter of the greatest interest to inquire in what part of the nervous system we must seek the psycho-physical processes.

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[1] *Cf.* "Zur Psychophysik der Gesichtsempfindungen," § 1. *Zeitschrift fur Psychologie*, Bd. X.

The conclusion that the nerve-fibres of the white matter are not the seats of the psycho-physical processes has been extended by analogy and with a high degree of probability to all nerve-fibres, *i.e.*, to all processes of nerve-cells, whether they occur in the white or grey matter of the brain or cord, and, since it was generally held until quite recently, that the nerve-cells and their processes constitute the whole of the essentially active parts of the nervous system, the conclusion that the bodies of the nerve-cells or of some of them are the seats of the psycho-physical processes seemed to be inevitable, and has in fact been very generally accepted, though its acceptance has been perhaps more often implied than expressed. It must be admitted that this view has remained singularly barren up to the present time.

With the advent of the "neurone-theory," however, a new possibility was opened up, for it is of the essence of this theory that there is no continuity of substance between the individual neurones, and it seems therefore necessary to assume that the process by which one neurone communicates its excitement to another may be, and probably is, different from that which constitutes the passage of the nervous impulse along axones or dendrites. If, then, we accept the neurone-theory, the problem of the seats of the psycho-physical processes presents itself in the form of a dilemma; they may be seated either in the bodies of nerve-cells or at the places of junction of neurones, at synapses.[1] In a paper published some years ago[2] I ventured to present certain arguments in favour of the second possibility, to urge that we must regard the synapses as the seats of the psycho-physical processes. At that time there seemed to exist no histological basis for this view, for it was commonly taught that, besides the nerve-cells and their processes, there exists in the grey matter nothing but fluid plasma and other substances not immediately concerned with neural processes, the blood-vessels and the neuroglia a purely supporting tissue. It seemed to me, therefore, and perhaps to others also, a view somewhat wildly revolutionary and hard to be believed. But since that time further reading has shown me that the idea is hovering, as it were, in the neurological atmosphere, and that new evidence pointing in the same direction continually appears. Further, a possible histological basis has been described by a high authority, while certain observations made during a study of visual sensations and in part already published[3] seem strongly to support the view.

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[1] Even if we incline to believe that the movements of all matter, or of all living matter, are associated with some kind of consciousness, we shall still have to admit that the fibres of the peripheral nerves and of the white matter of brain and cord are not immediately concerned with the higher kinds of consciousness.

As my previous paper was written primarily for psychologists the argument was based chiefly on psychological grounds and the physiological evidence was not detailed. It seems worth while therefore to attempt to hasten the precipitation of this view and to give it currency by putting together in this paper as briefly as possible all that can be said in its favour as against the view that the bodies of the nerve-cells are the seats of the psycho-physical processes.

First let me point out that neither view is necessarily materialistic, that neither excludes the conception of an immaterial active soul. If the soul is a valid conception, then we must admit of the soul that it is where it acts or is acted upon, so far as our ideas of space are in any sense applicable to it. And if the view that I advocate should prove to be true, then those who adopt the spiritualistic hypothesis will have to regard the synapses as the places of interaction of soul and body; as Lotze writes in the course of an admirable discussion of this subject: "Wherever there are elements with which the nature of the soul enables and compels it to interact, there it will be present and active; wherever there is no such summons to action, there it will not be or will appear not to be."[1] And, in order clearly to define the philosophical standpoint from which my enquiry sets out, I will quote another passage from the same work of this distinguished author: "We shall never succeed in analytically deducing the feeling from the nature of its physical excitant; we can only connect the two synthetically; and the physical event does not become a condition of the rise of the feeling until the sum of motions in which it consists meets with a subject which in its own nature has the peculiar capacity of producing feeling from itself. In this fact a limit is at once placed to all physiological and psychological enquiry. It is utterly fruitless to attempt to show how a physical nervous process gradually transforms itself (as we are told) into sensation or any other mental occurrence. There remains only the different but extremely important task of discovering *what* psychical event a and *what* physical stimulus a are as a matter of fact universally connected in the order of nature, and of finding the law by which a undergoes a definite change and becomes β, when a by a change equally definite (but definable only by a physical standard and not a psychical one) becomes b."[2]

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[1] The name proposed by Foster and Sherrington for the junctions of neurones without continuity of substance. (See "Sir Michael Foster's Text-book of Physiology," vol. 3, p. 929. Seventh edition).

[2] *Mind*, N. S, vol. vii. "A contribution towards an improvement in psychological method."

[3] *Mind*, vol. X. "Some new observations in support of Thomas Young's theory of light- and colour-vision."

The evidence seems to fall naturally under four heads, namely, the anatomical, the purely physiological, that derived from the study of sensations (the ground on which it is impossible to separate physiology and psychology), and lastly the psychological.

THE ANATOMICAL EVIDENCE.

As regards the structure of the bodies of nerve-cells, most histologists would seem to have returned now to the view of Max Schultze, that the fibrils, which seem to be the conducting elements of the axones, run into and through (in some cases at least) the cell-bodies and out into the dendrites.[1] And many of those who reject this view yet regard the "non-stainable substance" of the cell-body as closely allied to, if not identical in structure with, the substance of the axone.[2] The "stainable substance" of the perikaryon,[3] the tigroid bodies of v. Lenhoessek and other writers, is regarded by high authorities as nutritive in function. Nissl,[4] in particular, to whom our knowledge of this substance is chiefly due, has shown that when, through the action of poisons, it undergoes very marked changes, these changes are not directly correlated with the disturbances of nervous and psychical functions. It seems doubtful whether the non-stainable substance of the perikaryon contains any other substance than that which so closely resembles the conducting substance of the axones, but if any such third substance is present it must be in very small quantity only. Again, that the nutrition of the axones is in some way dependent upon the cell-body is fully established. This dependence is in all probability a special case of the general law established by the researches of Verworn and others on unicellular organisms, the law that an interchange of substance between the nucleus and the extranuclear protoplasm is essential to the continued activity and life of both.[5] Now in the case of most neurones the mass of the substance of the cell processes, axones and dendrites, is very great in proportion to the mass of the cell-body. Donaldson calculates that in some cases the volume of the axis-cylinder-process alone is as much as 187 times the volume of the cell-body. These considerations alone should make us hesitate to regard the cell-bodies as the seats of any other processes than those concerned in the conduction of the impulses and in the nutrition of the whole cell, and least of all can we assign to them the psycho-physical processes. For we must assume that these processes are the most complex and delicate of all that occur in the animal body, and for each primary sensation-quality at least we must assume a specifically differentiated psycho-physical process, involving in all probability complex chemical changes, and therefore a complex material substratum.[1] It may then be said with some confidence that there is no histological evidence of a sufficient material substratum for the psycho-physical processes within the cell bodies.

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[1] "Metaphysik," § 295. Bosanquet's translation. [2] *Ibid.*, § 238.

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[1] *Cf* Schaefer. Art. "Nerve cell," "Text-book of Physiology," p. 596. Edinburgh, 1900.

[2] *Cf.* L. F. Barker. "The nervous system and its constituent neurones," p. 186. London, 1900.

[3] The name suggested by Foster and Sherrington for the substance of the cell-body surrounding the nucleus.

[4] Nissl. "Nervzellen und graue Substanz" Part 1. *Munchener medizinische Wochenschrift*, Bd. 45, 1898.

[5] *Cf.* Max Verworn. "Allgemeine Physiologie." Jena, 3rd Ed., 1901.

How then does the matter stand with the synapses? As to the relation of the terminals of fibrils with those of other fibrils or with the cell-bodies at synapses, it was, until very recently, the generally accepted view that there is a simple apposition and no continuity of substance, and many interesting speculations have been advanced regarding the possible changes of relation of these terminals conditioning changes of functions.[2] This view of the lack of continuity was supposed to be true of all junctions of neurones, and its universal application seemed to be generally held as an essential part of the neurone-theory. But during the last few years doubt has been thrown upon this view. Continuity of substance has been proved to exist in certain instances, and with that tendency to generalise from a few particulars so curiously prevalent among histologists, some have concluded that the establishment of these instances of continuity deals the neurone-theory its death-blow. Of observations of this kind only the more important need be noticed. Dr. Alex. Hill has described a continuity of neurones by means of fine core-like threads stretching across from fibril to fibril and more or less sheathed in a stainable plasma, and he suggests that the relation of this plasma-sheath to the core is variable and conditions the degree of conductivity or intimacy of functional union between the neurones.[1]

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[1] For an interesting discussion of the probable nature of the psycho-physical processes see G. E. Müller, *loc cit.*, § 9.

[2] For a summary of these speculations and of the evidence for amœboid movements of terminals, see Duval "L'amoeboidisme du systeme nerveux." *Revue Scientifique.* Paris, 1898.

It is clear, of course, that if this kind of union be shown to be of general occurrence, the neurone-theory will not be weakened but rather strengthened, inasmuch as our conception of the relations of neurones to one another and of the constitution of the synapses will be clearer and more definite.

Held has shown that in the case of many of the cells in several of the nuclei of the pons, in the cortex of the cerebellum and in the anterior horns of the cord of the rabbit, it is possible to trace a growing intimacy of union between terminals of axones and cell-bodies. About the time of birth of the animal there is simple proximity of a group of fibrils, the terminals come into contact with and gradually fuse with the cell-body by a process that Held calls "concrescence." For some time the junction remains marked by a layer of more highly refractive substance, but in some cases this too disappears and the fibrils which may penetrate far into the cell-body can only be distinguished from it by their slightly different texture and staining-reactions.[2] Apathy has described in the ganglia of the leech and earthworm a state of affairs still more remote from the discontinuity usually postulated as the basis of the neurone-theory, namely, a fibrillar network passing without breach of continuity between and through the bodies of several nerve-cells.[3]

These observations by Held and Apathy seem to be not only not incompatible with the neurone-theory but rather to prove the existence of just such a state of affairs as is demanded by that theory when conceived in its only rational form. In the paper referred to above[4] I ventured to sum up the phylogeny of the nervous system as a process of constant proliferation of new neurones at the top of the system with a progressive integration of the older neurones of the lower (*i.e.*, older) levels into conduction-paths of increasing fixity, *i.e.*, of increasing intimacy of union of the constituent neurones, the intimacy of union being a function of the degree of organisation of the synapses. If there be any truth in this scheme we must expect to find, in the nervous system of any progressive species of vertebrate animal, all degrees of intimacy of union of neurones, from actual continuity in the cord and lower parts of the brain to mere proximity of terminals in the case of many synapses of the cerebral cortex, and we should expect to find a marked difference in this respect between the very young and the old animal.

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[1] A. Hill. "Note on thorns and a theory of the constitution of the grey matter." BRAIN, vol. xx., 1897.

[2] H. Held. "Beiträge zur Structur d. Nervenzellen." *Achr. für Anat. u. Phys.* 1897. *Anat. Abth.*, Heft 3 and 4.

[3] Mittheilung aus d. Zool., Station zu Neapel, Bd. 12, 1897.

[4] *Mind.* New series, vol. vii.

We should expect also to find a maximum degree of fixity of conduction-paths, of intimacy of union of neurones, in the nervous systems of some of the lowly and unprogressive types of invertebrate; for, as I pointed out, in the case of a lowly species that has followed for thousands of generations a simple mode of life in a simple environment, "it may be reasonably supposed that its nervous system is completely organised to carry out automatically its few motor reactions to the comparatively simple impressions made upon its sensory nerves."[1] Held's observations show us how, as a matter of fact, this phylogenetic process of organisation of neurones into fixed conduction-paths is recapitulated in the case of some at least of the neurones of the lower parts of the brain of the rabbit, while in the continuous networks running through cell-bodies in the ganglia of the leech, as described by Apathy, we may see conduction-paths of the highest degree of organisation, actual continuity of conducting substance implying a corresponding fixity of function. Unfortunately Held does not seem to have looked for concrescence between neurones of the cortex. It must be noted that Held does not regard his discoveries as in any way invalidating the neurone-theory, and that, at the end of his paper, he points out that the substance of the concrescence-surface is probably of high importance, that it is probably the part of any conduction-path most readily affected by poisons, and may play a part in the determination of sleep, fatigue and attention and of the variable lost time of reflexes.

The theory of the individuality and discontinuity of neurones must not, then, be held too rigidly. We must rather believe that while all are originally discontinuous they tend phylogenetically to become more and more intimately united into series to form conduction-paths of greater and greater fixity, a process that is recapitulated and carried further in the individual as the result of use.

And the neurone-theory must be modified in another important particular; we may no longer assume that the neurones, the cell-bodies and their processes, constitute the whole of the essentially nervous or active substance and that the only substances that lie between and around them are the blood-vessels, neuroglia, and lymph, merely supporting and nutritive in function.

For Nissl has recently brought forward positive evidence of a substance different from these, existing outside and between the neurones, a substance which is especially abundant in the cerebral cortex of man.[1] He writes: "the significance of the newer discoveries consists in this, that the nervous tissue may no longer be conceived as hitherto, as a community of numberless cell-individuals, but we must think of the nervous tissue as consisting of nerve-cells *and* a specific nervous substance, namely, the fibrillar substance This, if I am not mistaken, is to be conceived as a living substance that is derived from the protoplasm of the nerve-cells and doubtless stands to the nucleus-holding cell in a relation of intimate dependence, but which, in spite of that, must be also in other respects in a very considerable degree independent of the cells, as is shown by the fact that in great part it develops and maintains itself external to the cell. This fibrillar substance seems, according to the latest discoveries, to be the carrier of the nervous functions. It represents therefore the highest step of differentiation of the cell-plasma within the animal body." Nissl points out that this inter-cellular substance, and not, as is commonly assumed, the presence of many cell-bodies and dendrites, is the specific characteristic of the grey matter which distinguishes it from the white matter.

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[1] F. Nissl. *Loc. cit.*, three papers.

The principal evidence for the existence of this inter-cellular substance is that "the higher the grade of development of an animal is, the fewer nerve-cells inhabit an equally large volume of cortex"; and that in the human cortex it is impossible to account for the whole mass of the grey matter as nerve-cells and their processes and neuroglia, for, when the mass of these is subtracted, there remains, especially in the middle layers of the cortex, a very considerable mass of substance not accounted for. As to the structure or arrangement of this substance Nissl's statements are not very definite, but he inclines to regard it as fibrillar. Here then we have histological evidence of an abundant inter-cellular substance that may be regarded as the seat of the psycho-physical processes, or if anyone prefer the expression, as the material basis of consciousness, while in the case of the cell-bodies, as we have seen, there is no such evidence.

THE PHYSIOLOGICAL EVIDENCE.

We know that there are various states of the central nervous system characterised by differences in the degree of resistance offered to the propagation of the nervous impulses from the afferent to the efferent nerve-fibres, of which states sleep and waking are the most familiar. That the synapses are the seats of these changes in the degree of resistance has appeared probable to many writers.[1] We know also that, in passing through the central nervous system, the impulses are delayed, there is occasioned in some way what is known as the "lost time." Thirdly, we know that some part of the central conduction-path acts like a valve, permitting the passage of impulses from the afferent towards the efferent side, but preventing entirely, or at least offering great resistance to, passage in the reverse direction. Fourthly, we know that a very rapidly repeated or a continuous stimulus thrown into an afferent nerve or into the motor cortex issues in the efferent nerves as a regular series of impulses at the rate of about ten to nineteen per second, and that in voluntary effort also a similar series of impulses issues along the efferent nerves.

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[1] *Cf.* Duval. *Loc. cit.*

It is perhaps worth while briefly to resume the evidence that all these effects, the varying resistance, the lost time, the valve-like blocking of impulses, and the transformation of the continuous excitation, or rapidly repeated series of excitations, into a series of moderate rate, are determined at the synapses and not at the cell-bodies; for here again there seem to be only two possibilities—the problem appears as a dilemma—cell-body or synapsis.

In evidence against the cell-body we have the following facts: firstly, it seems to be established, as was noted above, that in many cases, if not in all, the fibrils of the cell-processes run through the cell-bodies without interruption, and there seems to be no direct evidence of any sort to show that the bodies of cells offer any resistance to the passage of impulses through them; we know on the other hand that in passing through a spinal ganglion the impulse suffers no appreciable time delay,[1] and that impulses, or at least electric changes such as always accompany a nerve-impulse, may be transmitted through the spinal ganglia in both directions;[2] lastly, we know from Bethe's remarkable and oft-quoted experiment on *Carcinus Mænas* that a nerve-fibre deprived of its cell-body may continue for some time to play its part in producing reflex movements in an apparently normal manner.

That the synapses are the seats of the valve-effect and are therefore at the root of what has been called the "law of forward-conduction of nerve-impulses," and that they occasion the lost time of reflexes and simple reactions, are propositions now pretty generally accepted. The former seems to be proved by the experiments of Gotch and Horsley, which showed that the nerve-impulse may be transmitted in the reverse of the normal direction through the spinal ganglia and through the efferent nerves, but is not, in the latter case, passed on to other neurones of the cord; while the latter is rendered highly probable by the simple process of excluding the alternative, the cell-body, no lost time being demonstrable in the case of the spinal ganglia. There is less agreement as to the seat of the determination of the rhythmic series of efferent impulses and the variations of resistance.

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[1] Schäfer. Art. "Nerve Cell," "Text-book of Physiol.," p. 604.

[2] Gotch and Horsley. *Phil. Trans.*, vol clxxxii. B.

Schafer, to whom we owe so much of our knowledge of the discontinuous rhythmic character of the efferent outflow, writes: "This rhythm must in all probability be set up in the motor nerve-cell," and he seeks to bring it under one point of view with the determination of the "lost time" by suggesting that every neurone has a latent period of excitation analogous to that of striped muscle-fibres.[1] But this hypothesis cannot, I think, be made to fit with the facts. The first difficulty that suggests itself is this—If the rhythmic discharge of efferent impulses at ten per second is to be explained by assuming a latent period of excitation for the neurones, we shall have to assume that the duration of this latent period is approximately 100 σ[2] for at least one neurone of the conduction-path concerned in the rhythmic discharge. For we cannot suppose that small latent periods of the individuals of a chain of neurones will be summed together to give the appearance of a long latent period in the last of the chain, except in the case of a single stimulus or the first of a series of stimuli; the period between any two impulses of a rhythmic series determined in this way could not be longer than the period of latency of that neurone of the chain which has the longest latency. Now a reflex contraction, or one produced by stimulating the cross-section of the cord, exhibits a rhythm similar to that of contractions determined from the cortex,[3] yet, as Windt has shown, the total lost time of a cord-reflex may be as small as 8 σ, and, as Schafer points out,[4] the lost time at each synapsis of a reflex conduction-path in the cord is probably only 2 σ. The latent period of the neurones of the cord, therefore, cannot be more than about 8 σ and probably not more than 4 σ, and therefore cannot account for a rhythmic interval of 100 σ.

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[1] *Cf.* Schafer on "The Block Between Nerve-cells." *Op. cit.*, p. 613.

[2] σ is the sign commonly used by psychologists to denote $\frac{1}{1000}$ sec., and will, I think, conduce to clearness if all brief periods are given in terms of σ

[3] Sherrington, "The Spinal Cord," "Schafer's Text-book of Physiol. p. 881.

[4] *Op. cit.*, p. 609.

The assumption of a latent period of excitation proves equally unsatisfactory when applied to the explanation of the lost time of reflex actions and of voluntary reactions. If such a latent period obtains, all analogy would lead us to expect that it should be approximately of the same duration for different neurones and for the same neurones at different times. But this is very far from being the case. In the first place, the lost time of a simple reflex is shorter the stronger the stimulus, and with a sufficiently strong stimulus may become inappreciable;[1] and in the case of simple reactions, too, the lost time is in some cases shorter with stronger stimulus.[2] Again, the delay at the synapses must be greater for most of the synapses of the cortex than for those that occur in the course of reflex conduction-paths of the cord. The lost time at a synapsis of the latter kind cannot be more than about 4 σ, perhaps not more than 2 σ,[3] yet the lost time of the cortex on direct stimulation of its surface is found to be about 20 σ, and this represents in all probability the lost time of a single synapsis, or at most of two. Again, the lost time of a single "motor" reaction may be exceeded by that of a simple "sensorial" reaction by 100 σ, and by that of a "choice" reaction by as much as 400 σ.[4] If, then we are to suppose the lost time at all synapses to be approximately equal we shall have to assume that in such a "choice" reaction the excitation runs through a chain of 100 to 200 neurones of the cortex successively, a conclusion that cannot be accepted for a moment. We must believe rather that in the reflex conduction-paths of the cord, paths of a high degree of constancy of function, the synapses are very thoroughly organised, *i.e.*, that their degree of resistance has been reduced to a minimum by frequent repetition of the particular reflex action in the individual and in the race, while in the higher parts of the nervous system the resistance, and therefore the loss of time, occasioned by the synapses is greater in the inverse order of their degree of organisation. This conclusion is borne out by the study of the effects of practice in shortening reaction-times. It is well known that the simple reaction-time may be somewhat shortened by practice until it becomes a purely automatic or reflex act, and in a similar manner reactions involving more complex mental processes, such as discrimination and choice, may on frequent repetition become more and more automatic in character, *i.e.*, they are performed more easily and regularly and with less and less clear conscious accompaniment, until they, too, resemble rather a reflex action, psychical activity being reduced to a minimum. And parallel with this progressive loss of psychic accompaniment goes a progressive shortening of the time lost at the synapses.

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[1] Rosenthal, quoted by Exner. *Entwurf z. physiol. Erklarung d. Psych. Erscheinungen*, pp. 48 and 55.

[2] Exner. Hermann's "*Handbuch d. Physiologie*," Bd. ii.

[3] Schäfer. *Loc. cit.*, p. 609.

[4] Külpe. "Outlines of Psychology," p. 419.

This shortening of the lost time at the synapses by repetition of the passage of the excitation across them may be best conceived as due to a diminution of the resistance offered by that delicate and complex inter-cellular substance, which, as it seems to me, we have to regard as the seat of the psycho-physical processes.

The consideration of how exactly the various and variable periods of lost time are determined I postpone to the concluding section of this paper, but will adduce here two lines of evidence bearing out the view that the principal factor is the degree of resistance offered by a specific inter-cellular substance. Such evidence is afforded by the consideration of the action of certain drugs. The observations of Langley[1] and of Langley and Anderson[2] on the effects produced by nicotine on nerves and ganglia seem to show that nicotine, in paralysing a sympathetic ganglion, acts upon the junctions of the fibrils with the cells of the ganglion, and this in far smaller quantities than will appreciably affect nerve-cell or fibre. This implies either a differentiation of the terminals of the fibrils or the existence of a specific inter-cellular substance peculiarly subject to the paralysing action of nicotine.[1] The junctions of nerve-fibres with the cells of glands and muscles are also peculiarly easily affected by drugs and by fatigue, and it is not improbable that the constitution of such junctions is similar to that of the junctions of the neurones

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[1] *Journ. of Phys.*, vol. xi.

[2] *Ibid.*, vol. xiii.

It is well known that ether, chloroform and alcohol, when applied directly to exposed nerves, will abolish their conductivity. But, if taken into the blood, they abolish the higher functions of the brain when present in such small quantities only as to leave the lower nervous mechanisms unaffected. I have elaborated this point in the paper before referred[2] to, and will therefore only briefly resume the argument as follows:—The functioning of the conduction-paths of the central nervous system seems to be abolished by an increasing quantity of any one of these drugs, strictly in the inverse order of the fixity of function of the paths, *i.e.*, of the degree of organisation of the synapses of the paths. Thus the extremely variable and complex and delicately-balanced processes involving self-consciousness are first diminished and then abolished; and these are followed in turn by the more stable, more habitual functions, *i.e.*, those subserved by relatively well-organised conduction-paths; while only in the deepest stages of the anaesthesia do we see affected the deep-seated vital reflexes, those whose conduction-paths, being constantly at work throughout life, have synapses of the highest degree of organisation. This is just the series of effects that must result if these drugs act first and chiefly upon the inter-cellular substance at the synapses, slowing or paralysing its metabolism in some degree and so raising its normal resistance. For where the resistance is normally greatest there complete blocking of the conduction-path must earliest result, and where it is least there conductivity will persist longest, the synapses will remain longest permeable to the nervous impulse.

The waste products of muscular activity exert an effect on the nervous system comparable in many ways to that of anæsthetic drugs such as alcohol and ether, and it seems probable that they too may act principally on the intercellular substance increasing the resistance of the synapses. Especially noteworthy is it that fatigue, like alcohol, prolongs the lost time of reactions.[1] But however this may be, there is evidence that the synaptic processes are peculiarly liable to become fatigued when any conduction-path is continuously stimulated. The evidence is of two kinds, the one kind, afforded by certain observations on the behaviour of visual sensations, belongs to the following section, the other may be dealt with at once, with the proviso that its full force cannot be made clear till the former also has been presented. In a recent paper Mr. A Levy[2] describes the fatigue effects that appear on repeatedly stimulating one spot on the motor cortex; the curve of contraction of the corresponding muscles being registered on a revolving drum. He finds that, when a faradic current is continuously applied, the contraction-curve of the muscle is maintained at a constant height for a period varying from 8 to 30 seconds and then falls suddenly, sometimes with a few clonic contractions, to the base line, and that then for a short period no contraction can be evoked even by a rather stronger stimulus applied to the same spot on the cortex. Levy writes: "It is difficult to regard this as evidence of anything else than that the motor cells have become fatigued."

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[1] Recent observations by Langley (*Journ. of Phys.*, vol. xxvi.) make this conclusion seem less certain, but by no means disprove it.

[2] *Mind*. N. S., vol. vii

Now we know that neither nerve-cells nor fibres in the peripheral system can be made to show any symptoms of fatigue at all comparable to this; and we know also that there are many reflex conduction-paths by means of which muscles may be kept in a state of contraction for very considerable periods with little or no signs of fatigue. A good instance of such long-continued activity of a reflex conduction-path is supplied by the carrying of a heavy pipe tightly clenched between the teeth while one remains unconscious of, or at least quite inattentive to, its presence. Now, between such a reflex conduction-path as is at work in this instance and one involving neurones of the cortex, the only differences, so far as we know, are firstly, that the former has by constant use become thoroughly organised so that the resistance of the synapses has been reduced to a minimum, while in the latter there must usually occur between cortical neurones synapses but little organised and of high resistance; secondly, that consciousness usually accompanies the activity of the paths of the latter kind, and not all, or only in some very obscure fashion, and in a minimal degree, the activity of paths of the former kind. It seems probable therefore that the sudden drop of the contraction-curve in Levy's experiments is due to fatigue, not of the motor-cells, but of the synaptic processes which effect the transmission of excitation from neurone to neurone, the resistance of the inter-cellular substance being so increased by this fatigue that the impulses set up in the superficial neurones of the cortex cease to be transmitted to the efferent neurones. Levy reports in the same paper other observations that support this view. He shows that with continued faradic stimulation, the sudden drop of the contraction-curve occurs earlier with a weak stimulus than with one of moderate strength, so that he is led to write: "It may then be concluded that cortical fatigue is more rapid in its progress as a result of weak stimulation." This, if true, would surely be a very extraordinary state of affairs; it is well-nigh incredible that any function should be fatigued more rapidly by a low than by a higher degree of activity. But if, as I suggest, the fatigue is synaptic and results in an increase of the normal resistance of the synapses, then it is obvious that the passage of a weak excitation may be barred by a smaller degree of fatigue, a smaller increase of the normal resistance than would be necessary to bar the passage of a stronger excitation.

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[1] Exner. *Hermann's Handbuch*, Bd. 2.

[2] *Journ. of Phys.*, vol. xxvi., "Fatigue of cerebral cortex," 1901.

Again, Levy shows that, if a faradic stimulation of about three seconds' duration be repeated at intervals of one second, the tops of the series of contraction-curves form a curve falling slowly and regularly and reaching the base line only after a very prolonged period of repeated stimulations. It appears, therefore, that the periods of rest of one second suffice to prevent the onset of that peculiar form of fatigue which, under continuous stimulation, causes the complete and sudden fall of the curve after from eight to thirty seconds; and that, when this form of fatigue is prevented, there manifests itself an effect just such as would result from a fatigue of the neurones, *i.e.*, from a diminishing energy of response to the stimulations of constant strength. It seems then probable that in these experiments we have to do with two kinds of fatigue, namely, fatigue of the neurones that manifests itself in a gradual diminution of the energy of response, and fatigue of the inter-cellular substance characterised by its rapid onset and still more rapid disappearance.[1]

THE EVIDENCE FROM THE STUDY OF SENSATIONS.

Certain phenomena that I have observed and in part described in a recent paper[2] seem to lend very strong support to the interpretation, suggested in the preceding section, of the fatigue-effects manifested by the motor cortex when directly stimulated. I find that if any dull or moderately bright patch of white or coloured light on a dark ground is fixated very steadily it is liable to undergo what I have called complete fading, *i.e.*, it will suddenly disappear from consciousness to reappear as suddenly and with undiminished brightness after an interval that varies from about half a second to several seconds. If the patch of light be upon a background that is not quite dark, but feebly illuminated, it happens not infrequently that the whole field of vision disappears, both the bright area and the background, leaving a sense of complete and extreme darkness, a darkness much more complete than is experienced on excluding all light from the eyes. This complete fading may occur during very steady fixation while both eyes are accommodated upon a near object, but it occurs far more readily if the accommodation be entirely relaxed and two similar bright images be combined by regarding them with the visual axes parallel And it occurs most readily when the patch of light is fixated by one eye only (the other being closed and covered) with relaxed accommodation and a + 4 D glass before the eye to preserve definition and perfectly steady fixation.[1] A noteworthy fact is that sometimes the fading is not simultaneous over the whole bright area, but proceeds from one side or other, so that a black shadow seems to sweep across the field and, sometimes, to retreat and advance several times before completely extinguishing the light.

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[1] V. Horsley (BRAIN, 1898) shows that if a continued faradic stimulus be thrown into the sciatic nerve, and the contraction curve of the reflexly excited crureus muscle of the same side be registered, the curve soon falls towards the base line If then the other sciatic be stimulated the muscle contracts rapidly and strongly again. Horsley concludes, therefore, that the fall of the contraction curve is the expression, not of fatigue of the reflex-centre, but of some effect allied to inhibition. I suggest that here again we have to do with synaptic fatigue, namely, at the synapsis by which the afferent fibres of the sciatic nerve make contact with the reflex-centre.

[2] "Some New Observations in Support of Thomas Young's Theory of Light- and Colour-vision," Section i. *Mind*, vol. x.

In the case of complete fading of a single patch of light on a dark ground, the coming and going of the image in consciousness seems somewhat arbitrary and lawless, but it is clear that the tendency to fade grows with the duration of fixation, and that it is far greater with relaxed than with accommodated eye, and with a dull than with a bright or very bright image. If, when a small white patch *a* has been fixated for three or four seconds only (the accommodation mechanism being relaxed), a second similar patch *b* is suddenly exposed so that the image of it falls on another part of the same retina, *or on a non-corresponding part of the other retina*, the image *a* merely exhibits some degree of loss of brightness; but if *a* is fixated for about fifteen seconds or more and *b* then suddenly exposed, *a* will usually disappear from consciousness at once, remain absent several seconds, and then return suddenly, appearing equally bright with *b*.[1]

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[1] Steady fixation is essential, as the slightest wavering of the fixation prevents complete fading or at once restores the image to consciousness if faded. To fixate steadily and at the same time to maintain the accommodation mechanism in a state of complete relaxation requires considerable practice. A method by which anyone may observe this curious phenomenon of complete fading without previous practice in the control of the eye-muscles is the following: Paste a sheet of white paper on a window-pane and fix flat against the paper a sheet of coarsely perforated zinc with circular or hexagonal holes from 1 cm. to 3 cm. in diameter; then fixate with one eye only a point about the centre of the sheet of zinc. The bright circles or hexagons may then be seen to pop in and out of consciousness in a very curious way, some three or four being usually absent from consciousness at any one moment. With relaxed accommodation and a + 4 D glass the effect may be observed still more readily.

In the paper on vision[2] I have described numerous instances of this inhibitory effect exerted by one visual image on another, and have pointed out its importance for the theory of vision, endeavouring to show, among other things, how it plays the chief part in all cases of simultaneous visual contrast. Here I am only concerned to show how the phenomena of complete fading and mutual inhibition of visual images lend support to the proposition that consciousness is determined at the synapses.

In the case of complete fading of a single image on a dark ground the light rays continue to effect in the retina the usual chemical changes,[3] and we are therefore compelled to assume that the excitation, set up by these chemical changes in the neurones of the retina, ceases to be propagated through the higher chain of neurones of the cortex in which consciousness is chiefly or wholly determined. And since, when the image returns to consciousness after an absence of one or more seconds, its brightness is not perceptibly less than before its disappearance, we cannot ascribe the failure of the excitation-process to pass through the higher neurones to any diminution of its energy. We are driven rather to suppose that at some point in the conduction-path the normal resistance to the passage of the excitation rises during continued activity until it reaches such a degree as completely to block the path, and that, during the short period of absence of the image from consciousness, this higher path being at rest quickly regains its former state, *i.e.*, the increased resistance rapidly falls again to such a point that the excitation passes through the path once more. Now, in the preceding sections of this paper we have already reviewed the evidence that points unmistakably to the synapses as the chief seats of resistance in any conduction-path and as the seats of the changes that produce variations in the degree of the resistance, and in the light of that evidence it appears in the highest degree probable that in the case of complete fading of visual images the increase of resistance is determined at the synapses. When, after fixating a patch of light *a* for about fifteen or more seconds, it is made to disappear from consciousness by throwing a similar image *b* on to another part of the same *or the other retina*, this can only be because in some way the rise of the image *b* to consciousness, or better, the passage of the excitation set up by it through the neurones of another part of the visual cortex, tends either to increase the resistance of the synapses of the conduction-path of *a* or to diminish the energy of the excitation-process seeking to pass by that path, which tendency is not powerful enough to prevent the passage of the excitation through the conduction-path of *a* unless the resistance of the synapses of that path has been raised by a previous period of activity of fifteen or more seconds. To discuss the nature of the inhibitory effect would carry me too far away from the subject in hand, and it is to the fatigue effect that I wish to draw particular attention, for on this depends the strongest strand of my argument.

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[1] For full descriptions of such cases see Observations x. and xi. in the paper referred to above. *Mind*, vol. x.

[2] *Ibid.*

[3] For proof of this see Observations vi., vii., and viii. *Loc. cit.*

For the occurrence of this increase of resistance of the cortical conduction-paths through fatigue of the synapses we may find abundant evidence, besides that of the kind adduced above, in the phenomena of the struggle of the two visual fields. It is a familiar fact that when we look at a bright white surface, with a red glass before one eye and a blue glass before the other, the surface usually appears alternately red and blue; there is an alternation of the red and the blue images in consciousness, *i.e.*, the blue image of one eye fades completely and the red image alone affects consciousness, until after a short period, that varies in length somewhat under different conditions, the red image fades completely and the blue one alone affects consciousness. For a description of a variety of cases and a discussion of binocular, and especially of monocular, struggle of colours, I may refer to my paper on vision.[1] The point on which I wish to insist here is that when the one coloured image disappears from consciousness, the neural events underlying that disappearance seem to be similar to those underlying the simple complete fading of a single visual image, or of one image inhibited by a second image on another part of the same or other retina; that is to say, the excitation ceases to pass through the neurones of the visual cortex, and the factor that determines the regular alternation of the two images is the rapidly oncoming and rapidly disappearing fatigue of the synapses of the cortex.

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[1] *Loc. cit.*

In support of this view we have the following facts:—(1) A very similar alternation in consciousness, *i.e.*, alternate predominance of an image and inhibition of it by another image, may occur in the case of two after-images formed on adjacent areas of one retina;[1] (2) if, when a red field is presented to one eye and a blue field to the corresponding area of the other eye, one eye be closed or covered for a brief period—one second will suffice—the colour presented to that eye always predominates over and inhibits the colour presented to the other eye as soon as the eye is uncovered, *i.e.*, the rested tract predominates over the relatively fatigued tract, even if the period of rest be not more than one second; (3) if by any one of several devices, as, for example, slight movements of objects in the field (the drawing of a hair across the field a little before the eye will suffice), the predominance of one field, say the red field, be prolonged, the blue field of the other eye tends more and more strongly to assert itself until, no matter how vigorous the movements in the red field, the blue predominates and the red field disappears from consciousness; all such devices merely direct attention to the one field, *i.e.*, they cause the excitation of the one cortical conduction-path to be re-enforced by the activity of higher levels, and we see that as the excitation continues to pass through this path the resistance of the path increases until, in spite of such re-enforcement, it yields to the inhibitory influence of the other rested tract; (4) if, accommodation being relaxed, a white field be presented to the right eye and a much less bright white or gray field to the corresponding area of the left eye, and if the left eye be covered over for some ten to fifteen seconds, while the right eye continues to fixate the brighter field, then on uncovering the left eye the bright image of the right eye yields to the much less bright image of the left eye, and disappears from consciousness for some seconds. I hold this last observation to be especially important, because it shows that the relative degree of fatigue of the conduction-paths is of more importance than the relative brightness of the two images in determining the predominance of one or other of them.

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[1] Observation xiii. *Loc. cit.*

The evidence of fatigue in the cortical conduction-paths thus afforded by the study of visual sensation corresponds in a striking manner with that yielded by Levy's experiments in direct stimulation of the cortex. Both kinds of evidence point to the occurrence in the cortex of a peculiar form of fatigue not assignable to the neurones. The sudden fall of the contraction-curve in Levy's experiments seems to correspond to the disappearance of the visual image from consciousness; the former occurs after continuous faradic stimulation for a period of from eight to thirty seconds, while the visual image shows no pronounced tendency to fade until after it has been present to consciousness for about ten or fifteen seconds. In both cases the failure of the cortical conduction-paths occurs earlier with a weak than with a strong stimulation. In both cases the fatigue-effect is very rapidly abolished during rest, and in both cases there is evidence that the energy of the excitation process is not diminished, or at least not diminished in such a degree as would account for its failure to pass through the cortical conduction-path. These two kinds of evidence, then, taken together and in conjunction with the abundant evidence from other sources pointing to the synapses as the chief seats of resistance in every conduction-path, a resistance variable in degree, enable us to assert with some confidence that the synapses of the cerebral cortex transmit the excitation from neurone to neurone by a process that is subject to a peculiar form of fatigue characterised by its rapid onset and rapid disappearance, and that this process, the material basis of which is presumably the intercellular substance, is far more readily fatigued than the processes of any other part of the conduction-paths. Now the psycho-physical processes must be of a very delicate and complex nature, in fact the most complex and delicately adjusted processes of the animal body, and for each primary sensation-quality at least there must be a perfectly specific psycho-physical process, probably in part at least of the nature of a chemical change. *Such a highly specialised process is just such as we should expect to be very readily fatigued, and it is therefore in the highest degree probable that the parts of a cortical conduction-path in which the psycho-physical processes occur are the parts most liable to fatigue. And we have good reason to believe that those most readily fatigued parts are the synapses.*

I will now bring together certain facts which indicate that the psycho-physical processes underlying our subjectively continuous psychical processes are discontinuous, *i.e.*, that they consist in the repetition at very brief intervals of a process that runs its course in a minute fraction of a second, the process of discharge of the afferent into the efferent neurone across the intercellular substance at the synapsis.

We know that series of stimuli applied at sufficiently short intervals to the terminations of the sensory nerve in the sense-organ will give rise to continuous sensations. Thus a regular series of air-waves falling upon the ear at a greater rate than about 16 per second gives rise to a continuous sensation of tone.[1] Touches or induction shocks repeated at the same spot of the skin may give rise to sensation of continuous contact when repeated about 36 times per second. In regard to visual stimuli the reports of different experimenters are not in agreement. Exner, by an ingenious experimental procedure and a somewhat questionable process of inference,[2] arrived at the conclusion that a visual sensation does not reach its maximum intensity until a considerable interval after light reaches the retina,

[1] If we may suppose that each phase of the complete wave acts on the nerve as a stimulus, the number of stimuli per second that gives continuous sensation will be about 32.

[2] Described by Helmholtz. *Physiol. Optik.*, p. 514.

in some cases nearly a quarter of a second; but the phenomenon known as Charpentier's bands seems to prove that a visual sensation may reach its maximum and decline again within one-sixtieth of a second after its inception. Exner gives about 44 σ as the interval that must separate two stimuli to the same retinal area if they are to be felt as two.[1]

Other observers, working with rapid alternations of light and darkness, generally with rotating sectors, have found that the rate or alternation at which fusion to continuous sensation occurs, varies with the colour and brightness of the light. Burch[2] has, however, pointed out that the duration of the intervals of darkness is of primary importance. Now Haycraft,[3] by a procedure that gave equal intervals of light and darkness, found that fusion occurred with the brightest light used at 19 alternations per second, which means a duration of the dark intervals of one thirty-eighth sec. (26 σ). This interval is just that at which successive touches may cease to give a discontinuous sensation (Exner and Preyer).

More important than these observations, which show that successive stimuli give rise to a continuous sensation when sufficiently rapidly repeated, are certain observations which show that a continuous stimulus may give rise to a discontinuous sensation, to a series of pulses of consciousness. Charpentier first observed that when a bright white surface is carried at a moderate speed across a dark field of view, its leading edge appears as a narrow band, that is brighter than the rest of the surface and is followed by an equally narrow dark band, and under favourable conditions he was able to observe as many as four or five alternating light and dark bands all about the same width, a width equal to the space through which the leading edge moves in about one-sixtieth second.[4] Shelford Bidwell confirms this observation and notes also that, when a single narrow band of light is carried across a dark field in a similar manner, it appears to him as four or five successive images separated from one another by narrow bands of darkness.[1]

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[1] Hermann's *Handbuch d. Physiol.* Bd. II.

[2] *Journ. of Phys.*, vol. xxi. "On Colour-Vision with Intermittent Light."

[3] *Journ. of Phys.*, vol. xxi. "Luminosity and Photometry"

[4] *Compt. Rend.*, 1887.

As to the appearances that are caused by a single brief exposure of the retina to a bright patch of light, and as to the phenomenon usually called the "recurrent image," the reports of different observers are strangely at variance and the whole subject is still in a very unsatisfactory state. The only fact on which all are agreed, I think, is that there occur rapid variations in the brightness of the sensation. My own observations agree most nearly with that one of Bidwell's mentioned above. This is not the place to describe them in detail or to attempt to correlate them with the various descriptions given by different observers. But since they afford evidence of the discontinuous character of the psycho-physical processes underlying continuous sensations they must be briefly mentioned. I have used various arrangements for securing the brief exposure of a patch of light, including one that ensures absolutely simultaneous appearance and disappearance of the light over the whole of the illuminated area. I find, however, that the slightest noise, such as is made by a rapid photographic shutter, renders quite impossible (for me, at least) the satisfactory observation of the brief visual image. I will describe therefore a series made by the following very simple method, noting that the other methods yield similar results, though less distinctly.

I obtained an oculist's trial-frame surrounded by opaque silk somewhat in the fashion of a motorist's goggles; the left aperture was covered in completely, the right one fitted with a metal disc with a circular hole 2 mm. in diameter at its centre, so that all light, save what reaches the right eye through this hole, is excluded. Wearing this arrangement I sit opposite to, and about three yards distant from, a vertical slit in the window-shutter of the dark-room, about 3 cm. in width and 10 cm. in length. In my right hand I hold a large sheet of black cardboard fashioned somewhat like a large Japanese fan, with wooden handle below, and in its upper part a vertical slit about 5 cm. in length and 1 cm. in breadth. By a small movement of the wrist this slit can then be carried rapidly across before the right eye and when the eye is directed towards the bright patch of light formed by the aperture in the window shutter, that is exposed to the eye for a fraction of a second and then immediately extinguished. On a bright winter's day, when the sky is a bright white surface, a sheet of coloured glass is put over the aperture in the shutter so that the eye, looking directly at the sky through the aperture, receives the image of a little slip of bright colour. When in this way a bright coloured image is allowed to fall on the retina for a moment, the character of the image is similar for all three colours used by me, namely, red, green and blue, and for a considerable range of duration of the exposure, from about one-fifth second downwards. The effect falls distinctly into two stages; the first stage consists in a pulsating or quivering image of the same colour as the glass used, declining rapidly in brightness, each pulse being less bright than its predecessor, until, after a period that increases with the brightness of the light from a small fraction of a second to nearly one and a half seconds, this first stage ceases and is replaced by a steady dull white after-image that dies away in the course of ten or more seconds. The pulses of colour are not separated by distinct intervals of darkness, rather each seems to begin before its predecessor has completely disappeared and the effect resembles the "coarse flicker" of of rotating sectors. The rate of the pulsation is slower in the image of a dull than of a brighter light, and it seems to decline with the brightness, so that, towards the end of this pulsating stage of the image of a light of moderate brightness the pulses can just be counted (which means that they are coming at about the rate of ten per second). At a time when the sun is opposite the window and shining through a thin white veil of haze in an otherwise clear sky I can, by repeating the observation with my eye in a series of positions such that it receives the light from parts of the sky successively nearer to the sun's disc, observe the effects of light of increasing brightness. With each of the three colours the effect is then similar ; with increase of brightness of the light the duration of the pulsating image increases and the pulsations become more rapid ; with light from the sky close to the sun's disc the effect resembles the "fine flicker" of rotating sectors ; and by choosing positions nearer and nearer to the direct ray of the sun it may be observed that the pulsating or quivering becomes so rapid as only to be noticeable when close attention is given to it ; and then, finally, when the direct rays of the sun fall on the eye, the bright image persists for some seconds as a continuous steady sensation, *i.e.*, without any perceptible quivering.

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[1] "Curiosities of Light and Sight," p. 191. London 1899; and *Proc. Roy. Soc.*, 1894.

This series of observations suggests very strongly that the momentary action of the bright light on the retina in some way overcharges the sensory neurones, so that they discharge themselves through the conduction-paths concerned by a rapid series of discharges of diminishing energy analogous to the oscillatory discharge of a Leyden jar, each discharge causing or constituting a psycho-physical process, or, as it may be called, a psycho-physical flash. And the transition from the slowly pulsating image through images of more and more rapid pulsation to an image that is perfectly and smoothly continuous, suggests that in the case of this last also the psycho-physical process underlying it is a series of such flashes very rapidly repeated, rather than a continuous process. If we accept this hypothesis provisionally it will lend itself well to the explanation of the effects of rapidly repeated stimulation of sensory nerves and of Charpentier's phenomenon, briefly reviewed above. We must suppose each effective stimulus applied to a sensory nerve, to give rise to at least one discharge, one psycho-physical flash. Thus in the case of sight and touch it would seem that when the stimulus, and therefore the psycho-physical flash, is repeated at shorter intervals than 26 σ, the resulting sensation is continuous in character, but discontinuous when the psycho-physical flash is repeated at greater intervals. In the case of Charpentier's phenomenon we must suppose that, under the conditions of the experiment, the moderately bright light is unable to cause the repetition of the psycho-physical flash at a greater rate than thirty times per second, *i.e.*, at intervals of about 33 σ, in the first moments after it begins to act on any given part of the retina; that is to say, each newly-stimulated part of the cerebro-retinal apparatus, being under the inhibitory influence of the image of the white surface formed on adjacent parts of the apparatus, exhibits a certain inertia, so that for a brief period of time the psycho-physical flash is repeated at greater intervals than 26 σ and the accompanying pulses of consciousness are felt individually. Bidwell's multiple images of the moving streak of light would represent again the successive psycho-physical flashes repeated at a greater interval than 26 σ.

THE PSYCHOLOGICAL EVIDENCE.

In this section I propose to recapitulate as shortly as possible the argument presented in support of my proposition in the paper before referred to.[1]

If, taking a general view of mental processes and directing our attention more especially to those involving clear or focal consciousness, we ask—What is the essential characteristic of mental activity? we find that it consists in the process of establishing in the mind relations between one thing and another. This process in its best-defined form is what is known as apperception, "the process by which a mental system appropriates a new element or otherwise receives a fresh determination."[2] The whole mind is shown to be organised into mental systems of very different degrees of complexity, corresponding to more or less complex objects;[3] each mental system is gradually built up by a series of apperceptive processes, each such process consisting in the presentation of some one aspect or feature of the whole object through some sense-organ, and the bringing of this feature into mental relation with various other aspects and features previously apperceived and incorporated into the mental system. "In almost every moment of waking life an apperceptive process is taking place; whenever an object is attended to the presentation of it is apperceived."[1] Mental activity, then, consists essentially in the perpetual succession of apperceptive processes, and the essence of apperception is the appropriation of the relatively novel presentation by the mental system built up by previous apperceptions, or, more shortly, is the bringing of the new into relation with the old, the product of past mental process. At each apperception of any given presentation of an object the appropriation of it by the mental system is more ready and more complete, while the consciousness excited by this aspect of the object becomes less and less vivid, until finally, when the appropriation of it by the mental system becomes complete, it is implicitly apprehended, or, in terms of conation, the stimulus supplied by this aspect of the object is responded to automatically, while some other aspect occupies the focus of consciousness.

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[1] *Mind*, vol. vii.

[2] G. F. Stout. "Analytic Psychology." The modern doctrine of noetic synthesis and the growth of mental systems by successive processes of apperception is admirably set forth in this work.

[3] The word "object" is used here in its widest sense.

When we attempt to translate all this into terms of neural process, our account must run in some such way as follows:—The mass of neurones constituting the nervous system is organised into functional groups and sub-groups, the members of each such group being intimately united together so that the excitement of any member of the group is shared by all the rest; such a group is the physiological counterpart of a mental system, or better, is the mental system in its physiological aspect. That an object is familiar to us means that, by successive presentations of its various aspects, the group of neurones directly excited by the presentation of any one aspect, has become intimately functionally related with the groups directly excited by the presentations of other aspects of the object. When we attend to any one aspect or feature we are apperceiving that feature, that is, we are relating it to other features, and this means in turn that the group of neurones directly excited by it is being brought into functional relation with those groups which have been previously organised by a similar process into the mental system, or complex group of neurones, corresponding to the total object. The physiological process constantly accompanying or underlying the focal consciousness of states of mental activity is then a process by which one group of neurones is brought into more intimate functional relation with another, generally a more complex, group of neurones. Now so far as we can at all conjecture, such an association of two groups of neurones means a change at the synapses which constitute the physiological boundary and points of contact between the neurones of the one group and those of the other (*i.e.*, physiological contact, not necessarily physical contact); a change of such a nature that thereafter any excitation can spread more readily across the synapses from one group to the other. Such a change, such a diminution of the resistance at synapses, seems to be the normal result of the passage of the excitation process across them.[1] Focal consciousness seems, then, to have as a constant physiological accompaniment the process by which the excitement of one group of neurones overcomes the resistance of a set of synapses and spreads across them to another group; and as these synapses become organised —*i.e.*, as their resistance becomes reduced more and more by repetition of the discharges across them—the consciousness accompanying the process becomes less and less clear and less intense until, when the synapses have become fully organised (*i.e.*, have their resistance reduced to a minimum), it is of a very obscure nature, forming at most only a part of what James[2] has called "the fringe of thought"; for the two groups of neurones have then become one functional group, one mental system, and a presentation, that formerly directly excited the one group only, is now implicitly apprehended and automatically reacted to, the excitation-process set up by it spreads through the whole mental system without having to overcome the resistance of unorganised synapses, and without arousing focal consciousness.

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[1] Stout. *Op. cit.*, vol. ii., p. 113.

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[1] Of this we have fairly direct evidence in the shortening of reaction times with repetition; but beyond this, it is no exaggeration to say that without this assumption it is impossible to begin to attempt to correlate psychic and neural functions or to form the vaguest conception of the physiological change underlying association and constituting the physical basis of memory.

[2] "Principles of Psychology."

The spinal cord and lower parts of the brain seem to consist wholly, with the exception of course of the long tracts of fibres merely passing through them, of completely organised groups of neurones, elementary mental systems, built up long ago in the history of the race by processes involving focal consciousness, and now merely the fixed reflex conduction-paths. While in the cortex, at the top of the whole nervous system, are neurones and groups of neurones not yet built up into mental systems, together with immensely complex mental systems capable of apperceiving a great variety of presentations, *i.e.*, of being brought in successive acts of attention into relation with various other groups of neurones, by the passage of the excitation-process across the synapses forming the physiological barriers between the groups.

The one physiological process directly correlated with focal consciousness is, then, this passage of the excitation across relatively unorganised synapses, and this would therefore seem to be, according to the terms of our definition, the psycho-physical process.

The same conclusion may be reached by looking at the facts from another point of view. The only objective evidence of the occurrence of consciousness is afforded by proof of retention by the nervous system. Where there is evidence of retention, of a functional change due to the activity of the nervous system, there we may conclude that consciousness accompanied that activity. Where any nervous activity proceeds automatically, *i.e.*, without consciousness, there we never find evidence of retention. Professor Bain[1] has pointed out this constant association of consciousness with those forms of neutral activity that lead to retention, and the same generalisation has been vigorously though somewhat grotesquely stated by Loeb in a recent work. He writes[2] that "so-called psychic phenomena appear, invariably, as a function of an elementary process, namely, the activity of the associative memory"; and again, "I think it can be shown that what the metaphysician calls consciousness are phenomena determined by the mechanism of associative memory." In the essay before referred to I have put the argument in the reverse and, as it seems to me, the correct order, namely, that what the plain man calls consciousness determines those effects which Prof. Loeb attributes to the activity of a metaphysical entity, namely, the associative memory. But, whatever way the truth may lie as to the causal relation, the important point for the purpose in hand is that the two things are constantly together; wherever there is memory or other evidence of retention, consciousness accompanied, and probably was one of, the conditioning factors of that retention.

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[1] *Mind*, N.S., vol. iii.

[2] "Comparative Physiology of the Brain and Psychology." London, 1901, pp. 213-214.

[3] *Mind*, vol. vii.

Now retention means the persistence of some physical change in the conduction-paths. And the persistent change which is the physical basis of memory or retention can only be conceived as consisting in such a union of the units composing a conduction-path that they shall in future tend to be simultaneously active, and this must mean an increased intimacy of connection between neurones, the result of organisation of the synapses through which they act upon one another. Similarly, the physical basis of that form of retention known as the association of two ideas by contiguity in time can only be conceived as an organisation of, a diminution in resistance of, the synapses through which the two conduction-paths, whose activity is the physical basis of the two ideas, can act upon one another. Such organisation of synapses is, as we have seen reason to believe, the result of the passage of the excitation process across them, which passage across unorganised synapses is then the only physiological process known to be constantly accompanied by consciousness, and therefore in all probability constitutes or immediately determines the psycho-physical process.

THE APPLICATION OF THE CONCLUSION ARRIVED AT.

There is, then, a considerable mass of evidence, histological, physiological and psychological, pointing to the synapses, more especially those of the cerebral cortex, as the seats of the psycho-physical processes, while, so far as I can discover, there is no reason of any kind whatever to suppose that the cell-bodies are the seats of those processes, as has been so commonly assumed. No one piece of this evidence, taken alone, is of a kind to force upon us the acceptance of this view, and from the nature of the case we cannot hope that any such direct and unmistakable evidence will ever be forthcoming. In this final section I wish to show that the conclusion arrived at is not one of theoretical interest only, but is such as to lead us on to new and fruitful conceptions as to the workings of the nervous system.

In a recently published pamphlet[1] von Kries has pointed out that, in spite of the immense progress made of late years by our knowledge of the structure of the nervous system, our knowledge of its functions seems by comparison to have remained almost stationary, owing to the lack of new conceptions distinctly physiological, not merely anatomical, in character. Von Kries believes that this is due to an unwarrantable acceptance of what he calls the conduction-hypothesis (Leitungsprinzip), the hypothesis that all nervous activity consists in the propagation of some state or process of excitation from part to part, such as we observe in the peripheral nerves, the nature or quality of the process varying according to the properties of the conduction-paths. He argues that this conduction-hypothesis has proved itself barren and that it is incapable of enabling us to describe even in vaguest outline the more complex functions of the brain, and he attempts therefore to give new vitality and increased definiteness to the hypothesis of the individual functioning of the ganglion-cells, the view that each cell is a psychic unit capable of determining within itself a more or less complex state of consciousness.

It does not seem to me that v. Kries makes out a sound case against the conduction-hypothesis, and the alternative that he proposes would seem to stand towards neurology in a relation similar to that of the doctrine of vitalism towards physiology in general, and to be therefore rejected so long as there is any hope of progress along other lines. But it must be admitted, I think, that, if the conduction-hypothesis is to remain our fundamental conception of nervous process, it sadly needs to be refined and fertilised by supplementary hypotheses. Towards the formation of such a hypothesis, a distinctly physiological conception, I hope to contribute my mite by drawing in rough outline a scheme that shall bring under one point of view the various facts dealt with above, together with the conclusion arrived at as to the functions of the synapses. And first, I would venture to urge that physiologists ought not to hold themselves slavishly dependent on the physicists, but that they must press on towards the solution of their own problems, building up for themselves such conceptions as shall enable them best to describe the peculiar processes that they have to study. Nor, in forming such conceptions, should they allow themselves to be hampered by a too great desire for formal consistency, but should follow the example of the physicists in this respect. The most wonderful achievement of our time is perhaps the command over, and insight into, electrical processes acquired by physicists, yet it would seem to be still impossible to describe the phenomena consistently in terms of any one of the several hypotheses as to the nature of electricity; again, the most fruitful hypothesis ever developed is perhaps that of an ethereal medium to which an impossible and inconceivable combination of properties is assigned by physicists.

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[1] *Ueber die materiellen Grundlagen der Bewusstseins-Erscheinungen*, Mohr, Tubingen, 1901.

We have seen above that Schäfer's suggestion of a latent period of excitation of each neurone cannot be satisfactorily applied to the explanation of the "block" between neurones and of the rhythmic discharge of efferent impulses. Another suggestion recently put forward by Professor Richet[1] must be briefly mentioned. Richet seeks to explain the phenomena of the retardation of the excitation-process in the central nervous system and the rhythmic efferent discharge by attributing to every nerve-cell a refractory period of nearly 100 σ following the passage of each impulse through it. He regards neural activity as consisting in a series of diphasic waves, of which the negative phase constitutes the refractory period of the nerve-cell. I do not know that there is any one fact that will suffice to disprove this view outright, but we may note in the first place that Richet offers us, as proof of the wave-character of the excitation-process, merely the fact that it can be diagrammatically represented as a wave. In a very similar manner we may represent a man's appetite for food and drink, or the war-like feeling of the country, and many other things, as diphasic waves of which the negative phase corresponds to a refractory period; but to do this is merely to speak in terms of analogy, and by no means proves that these happenings consist of wave-movement in a physical medium. Further, we know that a nerve-fibre will respond to each one of a rapid series of stimuli, and that if it has any refractory period its duration must be less than 1 σ,[1] while Charpentier's phenomenon seems to prove that the psycho-physical process may be repeated much more frequently than ten times per second, at intervals certainly not greater than 30 σ. This view, too, would seem to offer little promise of leading us to an effective method of describing the workings of the nervous system, and to be in this respect much inferior to the one that must now be dealt with.

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[1] Address to Brit. Assoc., Dover, 1898, reported in *Nature*.

Many considerations indicate that, in the workings of the nervous system, an important part is played by the charging of the individual neurones with an undefined something, and that a certain potential of charge must be given to any neurone before it can communicate its excitement to others, *i.e.*, before the charge can overcome the resistance offered by the synapses.

Exner recognises the necessity of adopting some such conception when he writes: "The reflex contraction in reply to a momentary stimulus has always a course very different to that of a contraction evoked by direct stimulation of the centrifugal nerve. It lasts longer, and may prolong itself to a tetanus. That indicates that somewhere in the centre (centrum), let us suppose in the end-twigs or in the ganglion-cells, a sort of charging (Ladung) takes place under the influence of the stimulus, and that this charge of the centre discharges itself through the outflow of the excitation to the muscle."[1] And throughout this work this idea of a "Ladung" of centres is frequently introduced. Bechterew[2] has given expression to an hypothesis of this kind, suggesting very roughly an analogy between the chain of neurones and a chain of Leyden jars. In an important and interesting essay Goldscheider[3] recognises the need of some such conception, and propounds the doctrine of the "neurone-threshold," the importance of which for therapeutics and pathology he insists upon. He recognises "that the principal conduction-resistance is offered by the passage (Uebertritt) of the excitation from neurone to neurone" (p. 5), and that the varying degrees of this resistance account for varying periods of lost time in the same and in different conduction-paths. "We must attribute a certain inertia (Trägheit) to the process that intervenes between the neurones (dem zwischen den Neuronen sich abspielenden Vorgange); otherwise we cannot understand the isolated conduction through the labyrinth of neurones in the gray matter" (p. 9). He admits the possibility that this resistance may be conditioned by an inter-cellular substance, but he thinks it improbable, and he attributes both the facilitation and organisation of conduction-paths (Exner's "Bahnung" and "Ausschleifung") to the neurones. While recognising the importance of this doctrine of the neurone-threshold and the enlightenment that it brings us, I venture to think that we shall receive still greater enlightenment if, accepting the indications afforded by all the different kinds of evidence adduced in the preceding sections of this paper, we assign the principal resistance in all conduction-paths to the intercellular substance at the synapses and so convert the doctrine of neurone-thresholds into one of synapsis-thresholds. The adoption of the threshold hypothesis in this form brings with it at once a great and obvious advantage. It introduces a great refinement into our conception of the mechanism that determines the course of any excitation in passing through the labyrinth of paths from afferent to efferent nerves. In place of regarding each neurone as having a certain threshold common to all its dendrites, each pair of fibrils that meet in a synapsis will be regarded as separated by an inter-cellular substance whose degree of resistance or threshold has been determined by the history of that synapsis in the individual and the species, and our conception of the mechanism that determines the course of any excitation will then be refined in proportion as synapses are more numerous than neurones.

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[1] *Cf.* F. Gotch, Art. "Nerve," p. 475 in Schäfer's "Text-book of Physiology."

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[1] *Entwurf zu einer physiologischen Erklärung der psychischen Erscheinungen*, p. 49. Wien, 1894.

[2] *Neurolog. Centralblatt*, 1896.

[3] *Die Bedeutung der Reize im Lichte der Neuron-Lehre.* Leipzig, 1898.

The idea of a certain charging of neurones has then been entertained by authors of high authority, but I have not been able to discover that anything has been said as to the nature of that something with which the neurones are supposed to be charged, although an effective working conception of its nature must be of the highest importance if there be any truth in this view. I think that for the present it may be best conceived as a fluid, and I propose that this fluid shall be called "neurin."[1] It might of course be called the nervous fluid, or nervous energy, or "animal-spirits," or a very subtle ether, but the name I suggest is preferable because it implies nothing beyond the fact that the thing named has to do with nerves. I have little doubt that at some future date it will be regarded as some form of motion of some species of ether, or of some still more remote hypothetical medium. But just as the fluid-theory of heat, the two-fluid-theory of electricity and the corpuscular theory of light furnished probably the most useful working conceptions for the sciences of heat, electricity and light at certain stages of their development, so neurin may, I think, be most usefully conceived as a fluid in the present state of neurology, and I think it would be unwise to attempt to regard it as a variety of any one of the forms of energy known outside the animal body, although it is easy to discover points of resemblance to both electricity and magnetism.[1] As Barker writes: "It is by no means impossible that in the nervous system forms of energy are concerned which do not exist outside the animal body and which yet remain to be recognised and studied;"[2] and we may perhaps go even further than this and say that the existence of some such form or forms of energy may be assumed with a very high degree of probability; for since, so far as we know, consciousness is only associated with transformation of energy within the living animal body, the forms of energy concerned must be of a highly special and peculiar nature.

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[1] I am indebted for the suggestion of this word, the adjective form of *νευρον*, to my friend Prof. G. C. Moore Smith.

I will therefore sketch in rough outline a scheme of the part that the fluid neurin seems to play in the workings of the nervous system. In virtue of its normal vital activity every neurone continually produces neurin in small quantity; and the neurones connected with the sense-organs and surface of the body generally are almost perpetually played upon by feeble stimuli that excite them to the production of neurin in rather larger quantities. Neurin tends always to flow from a place of high potential to places of lower potential. The neurin so produced therefore tends continually to flow from afferent to efferent neurones, in virtue of the higher potential of the sensory neurones and of the valve-like nature of the synapses, passing across the synapses by a sort of leakage, and so escaping by the efferent nerves into the muscles as a continuous gentle stream, maintaining that state of continued gentle contraction of the muscles which we call their tone.[3] It is possible that the slow leakage across synapses is the basis of that obscure affection of consciousness which has been called the "general sensibility" and forms the groundwork of the psychological self.

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[1] I do not mean to imply that the numerous attempts that have been and are being made to exhibit the nervous impulse as an electrical phenomena should not be regarded with the fullest approval. I mean that we ought not to wait for the final issue of such attempts before endeavouring to define more fully the nature of neural processes.

[2] "The Nervous System and its Constituent Neurones," p. 249.

[3] In this connection it should be remembered that in spite of the oft-repeated statement to the contrary effect, a continuous current of constant strength is capable of bringing about a continued contraction when thrown into a muscle-nerve preparation under suitable conditions. *Cf.* Biedermann "Beiträge z. Kenntnis de Reflex Function des Ruckenmarkes," p. 410, *Pflugers Arch.*, Bd. 80.

When any adequate stimulus falls upon a sense-organ the sensory neurones affected by it are excited to the production of much larger quantities of neurin per unit of time. Let us take the case of a ray of light falling upon the retina and there setting free a substance that chemically stimulates the nerve-endings, and let us call the neurones forming a conduction-path leading from the sense-organ to any motor neurone, *a*, *b*, *c*, *d*, &c., each one, as *b*, being efferent to its predecessor in the chain *a* and afferent to its successor *c*. Then *a*, continuously stimulated by the exciting substance in the retina, produces neurin far more rapidly than it can escape by leakage into *b*, so that in a small fraction of a second the potential of the charge in *a* will rise to the level of the threshold of the synapsis *a*—*b*. A sudden discharge of neurin then takes place across the synapsis from *a* to *b*, and its sudden arrival in *b* acts upon it as a stimulus to the rapid production of more neurin, so that *b* then has a double charge of neurin and therefore rapidly discharges into *c*; and so the process of discharge and stimulation of afferent into efferent neurone is repeated, until the last of the chain discharges itself into the muscle and brings about contraction. To go back now to the sensory neurone *a*; as it discharges, the exciting substance in the retina continues to stimulate it to rapid production of neurin, and therefore, in a very short time after the first discharge, the potential of its charge rises again to discharging point, and so once more the whole chain of neurones is charged, stimulated and discharged in turn, and a second impulse flows out to the muscle, and so on again and again. It is necessary to assume that the neurones at the efferent end of the chain have a larger capacity for neurin than those of the afferent, or of the central parts, so that, while the latter discharge many times a second (more than thirty-six times per second as we have seen in case of a continuous visual sensation), the large motor neurones of large capacity have their potential raised to discharging point less rapidly and therefore discharge at a slower rate—about 10-20 times per second, according to the intensity of stimulus and other conditions. And the conduction-path excited is probably never so simple as suggested above, there is probably always some spreading out of the path, so that for each sensory neurone stimulated several neurones towards the efferent side are affected, and the more intense the stimulus the greater the spread of conduction-path involved.[1] For with the minimal effective stimulus neurone *a* will only discharge by the synapsis of lowest threshold into perhaps one *b* neurone only, while with stronger stimulus the potential of *a* may be raised so rapidly and to so high a point that discharges take place by synapses of higher threshold into other *b* neurones. And this may be repeated in the case of each of the *b* neurones and of the neurones of the succeeding levels; so that, as we know, if the stimulus be a very strong one and the general state one of high excitability, almost all the muscles of the body may be thrown into activity.

This view of the charging and discharging of neurones not only brings under one point of view the loss of time at the synapses and the rhythmic discharge in the efferent nerves, but is supported and, we may almost say, necessitated by an array of facts that find a ready explanation through it. Of these the chief are certain peculiarities of the reflex response, the phenomena of summation of stimuli and of facilitation (Bahnung), and certain facts which prove the existence of a state of residual excitement or rather the persistence of residual charges of neurin after the passage of an excitation.

"There is a distinct relation between the intensity of the external stimulus and the latent time of spinal reaction, the latter becoming much shorter as the former is increased,"[2] and the lost time may even become inappreciably short.[3] The more powerful stimulus leads to a more rapid production of neurin, so that the potential of the sensory neurones sooner passes the synapsis-threshold.

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[1] Probably there is usually a spreading out in the middle parts of a conduction-path, and some reconcentration on the motor side.

[2] Sherrington. "The Spinal Cord," Schafer's "Text-book," p. 829.

[3] Rosenthal, quoted by Exner. *Entwurf, &c.*, p. 55.

"A single induction shock, the discharge of a Leyden jar, a single prick, &c., can excite a reflex action. Such brief 'single' stimuli must, however, in order to produce reaction, be violent, so violent as to injure the sensifacient tissue too much for repetition. Even when thus strong they are less efficient than less severe stimuli applied not singly but repeatedly."[1] The effect of a stimulus is to hasten during its incidence the normal slow production of neurin, rather than to give rise to an explosive setting free of energy, hence the importance of the duration of the stimulus.

"The reflex arc, as judged by the contraction of its muscles, behaves somewhat like cardiac muscle in responding its approximate best, or not at all. In other words, internal conditions affect the amount of response much more than does mere intensity of external stimulus."[2] In the simple conduction-path of a spinal reflex the resistances of the well-organised synapses are a comparatively fixed quantity; hence the amount of the charge and discharge of neurin is approximately constant.

"Stimuli far too weak to singly evoke reflex action, are easily able to do so if repeated, with separate electrical or mechanical stimuli the reflex tends to appear after a given number of stimuli have been applied, although the time intervals between the stimuli may be varied within wide limits—*e.g.*, ranging between ·05 sec. and ·4 sec."[3] Each stimulus causes the generation of a quantity of neurin in the sensory neurone too small to raise its potential to the synapsis-threshold, but, when the stimuli are repeated at such a rate that each fresh quantity is formed before any considerable fraction of the preceding charge has leaked away, the charges accumulate until the potential reaches the synapsis-threshold. In such cases the failure to discharge may perhaps occur, not in the sensory neurones, but in the efferent neurones of large capacity. A similar summation of stimuli is illustrated by the cough that follows prolonged slight irritation of the throat (Exner). Urbantschitsch[4] has shown that the note of a very gently-sounding tuning fork may fail to be heard until it has fallen on the ear for one or more seconds. This would seem to be a case of summation of stimuli entirely comparable to the foregoing cases.

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[1] Sherrington. *Loc. cit.*, p. 828.

[2] Sherrington. *Loc. cit.*, p 829.

[3] Sherrington. *Loc. cit*, p. 828, quoting James Ward.

[4] Quoted by Exner. *Entwurf. z. einer physiol. Erklärung, &c.*

Exner has shown[1] that if a spot of skin and a spot on the motor cortex be found, stimulation of which leads to contraction of the same muscle in both cases, then weak stimuli applied to these two spots re-enforce one another whether they be separately sub-minimal or not, and whether they be simultaneous or successive at any interval less than three seconds. This is the typical instance of what Exner has called "Bahnung," a word translated by Sherrington as "facilitation." Exner himself regards summation of stimuli as but a special case of "Bahnung," and the explanation in terms of neurin would seem to be entirely similar; in the case described the two conduction-paths, that from the spot on the skin and that from the cortex, converge at some point in their course to become one, and in this part, whose neurones are common to both paths, accumulation of charges of neurin takes place, whether through simultaneous charging from both directions or through summation of successive charges.

Exner[2] has pointed out that the preparation for a reaction, effected by expectant attention, consists in a charging of the conduction-path concerned, and is a process essentially similar to the facilitation of the paths in the last-mentioned experiment. We must suppose that, in response to the premonitory signal, the neurones of the path are charged with neurin almost up to the synapsis-thresholds, so that, when the expected stimulus falls on the sensory neurone, it has to increase the charge of neurin in it but very slightly to lead to its discharge, and so to the successive discharge of the whole chain of neurones constituting the conduction-path to the muscles. As Exner has pointed out, there is no further act of will involved in this response to the stimulus; the whole process takes place automatically, and may be brought about by any sudden sensory stimulus, and, what is particularly significant, it may occur after the subject has ceased to intend to react.

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[1] *Op. cit.*, p. 78.

[2] *Op. cit.*, pp. 158 and 131.

The phenomena of facilitation and the summation of stimuli afford evidence of the persistence in neurones of a charge of neurin for some seconds, and there is other evidence pointing to the persistence of a residual charge after the discharge of neurones. In the case of the cord this is indicated by experiments which prove that the production of a reflex is rendered easier by an immediately preceding activity of the conduction-path concerned.[1] Similar evidence in the case of neurones of the brain is afforded by certain psychological observations. In an important paper recently published,[2] G. E. Müller and Pilzecker have shown that an image (they dealt chiefly with nonsense-syllables) that has occupied consciousness tends to rise again to consciousness spontaneously, as it were, *i.e.*, without any assignable reproducing association, a tendency to which they give the name "Perseverations-tendenz"; and they show that this tendency is strong at first but declines rapidly, so that after a few minutes it but rarely makes itself felt. And they have further shown that during a similar period, following the succession of two images in consciousness, the association between those two images continues to increase in strength. My suggestion is, that there remains in the group of central neurones concerned in the production of an image a residual charge of neurin, so that their potential remains not far below the discharging point, and a very slight stimulus will cause their discharge, and so the revival of the image in consciousness; and that the gradual escape of this residual charge, by leakage across synapses, is the cause of the continued diminution of resistance of the synapses, *i.e.*, of increasing fixity of the association. The same authors show that the time needed for the reproduction of an image by an associated image is much shorter immediately after the image has passed from consciousness than a few minutes later, while the certainty of the association is not correspondingly greater. This result also points to the persistence of a residual charge identical with that to which, as I suggest, the "Perseverations-tendenz" is due.

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[1] Sherrington. *Loc. cit.*, several experiments, p 842

[2] Muller and Pilzecker. "Experimentelle Beiträge zur Lehre vom Gedächtniss," *Zeitsch. für Psychologie*, Ergänzungsband i. 1900. Summary in *Mind*, vol. x

Other evidence of the residual charge is afforded by the "primary memory-image." This is the name given to the memory-image of an object during a short period immediately succeeding its presentation through any sense-organ. It is found that during the first few seconds the image can be revived by a voluntary effort of attention with peculiar vividness and accuracy, that "after ten seconds a considerable waning has taken place, and after 100 seconds all that is distinctive of the primary image has probably ceased."[1]

Of these various cases that are so readily described in terms of neurin, those which indicate the persistence of a residual charge seem to me the most important, for they show that the activity of the neurones cannot be regarded as a mere propagation and augmentation of an impulse, as is commonly assumed to be the case in peripheral nerve-fibres; or as a wave movement of some unknown medium that sweeps through them, as Richet suggests; but that we must assume that there is generated in the neurones something comparable to a charge of heat or of statical electricity that can be stored up for a time and can leak slowly away.

If, however, the "primary memory-image" and the "Perseverations-tendenz" and allied facts are to be explained as due to a residual charge of neurin, we have to assume that some part at least of the tracts concerned in the production of a sensation and in the reproduction of that sensation in memory are identical. This is a point that has been much disputed. Bain and others have upheld the view of Hume, that the memory-image differs from the sensation only in being less vivid or intense, and they assume therefore that the neural elements concerned are identical. There are, however, few psychologists who would now maintain this view in all its original simplicity. Ward has put together the evidence that proves that the seats of the sensation and of the memory-image of it are not identical, and he concludes that, "In the complete or apperceived presentation, as in the complete or overt action, we must suppose, then, that the subcortical centres contribute whatever constitutes reality or actuality, of which intensity is one but by no means the sole characteristic."[1] I venture to think that, though this statement comes very near the truth, the view of cerebral activity that I am suggesting will enable us to put the matter in a clearer light, which I will indicate as briefly and as simply as possible. Let the cortical conduction-path concerned in the production of the sensation be schematically represented as simply as possible by a chain of three neurones, *a*, *b* and *c*, afferent, central and efferent respectively. Then, on stimulation of the sense-organ, the excitation spreads through the lower afferent neurones up to *a* and through the chain of cortical neurones, *a*, *b*, *c*, and so out to some motor centre; and the discharges across the synapses *a*—*b* and *b*—*c* constitute the psycho-physical processes that determine the sensation. Now when by association the idea of the same object is reproduced as a memory-image, the neurone *b* is excited not from *a*, but through some neurone of its own level, and it discharges through synapsis *b*—*c* as before. We may then suppose that the psycho-physical processes at synapses *a*—*b* and *b*—*c* are identical in quality and that the peculiar and undefinable quality that distinguishes the sensation from the idea, the presentation from the representation, is due simply to the duplication of the psycho-physical process; or more probably that, while the processes at *a*—*b* and *b*—*c* are essentially similar in quality, they yet differ in some respect, probably in that the discharge of neurin at *a*—*b* takes place at a higher potential than the discharge at *b*—*c*.

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[1] J. Ward, art., "Psychology," *Encycl. Brit.*, 9th ed.

In this way we may understand why it is that, while there are so many urgent grounds for regarding the seat of the sensation and of the idea as identical, yet on other grounds such a view is shown to be untenable; there is partial identity of "locus," the "locus" of the idea is a part only of the "locus" of the sensation.

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[1] "Assimilation and Association," *Mind*, N.S., vols. ii. and iii.

This scheme lends itself so well to the description of the neural events underlying mental processes that I am tempted to go on indefinitely, but I must content myself with the exposition of one other important point. We must believe that the presence of a charge of neurin in a neurone, even while its potential is below the threshold of the synapsis, tends in some way to lower the resistance of the synapsis, it may be that the terminal is protruded so as to diminish the the thickness of inter-cellular substance, or, more probably, that some change of state is induced in that substance; suppose, for example, that the charge of the neurone were a charge of heat, and that rise of temperature diminished the resistance of the inter-cellular substance, the effect of an infra-liminal charge would then be parallel to that produced by a charge of neurin. And the facts of sensory attention, especially that aspect of them insisted upon by G. E. Müller,[1] indicate that this effect is probably exerted on both the afferent and the efferent synapses of a neurone; to go back to the scheme of the preceding paragraph, a charge given to *b* from neurones of its own level lowers the resistance of the synapsis *a*—*b* as well as of *b*—*c*; and this effect seems to be in fact an essential part of the facilitation-process. Experimental evidence of the truth of this view is afforded by an experiment carried out by Grossman under Exner's direction, and I must refer to the latter's account of it.[2] The essence of it was, however, to show that, when of three nuclei which normally work together sending out rhythmic series of impulses one is isolated, the other two continue to discharge, but with a slower rhythm and with an increased energy at each discharge; so that, as Exner writes, "We see therefore that the phenomenon of 'Bahnung' may consist not only in charging of centres, but also in the lowering of the discharge threshold."

I will bring this paper to an end by pointing out how the conception of neurin may be applied to the explanation of certain changes in the general state of the nervous system.

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[1] *Zur Theorie der sinnlichen Aufmerksamkeit.* 1873.
[2] *Op. cit.*, p. 80.

The increase of the reflex activity of the cord that ensues when it is separated from the brain, may be explained as follows:—In the normal state a part of the neurin, generated by the sensory neurones leading to the cord under the influence of the gentle stimuli perpetually acting upon them, is led off by the ascending tracts into the brain and slowly filters through it, keeping up its tone without leading to discharges. When the brain is cut off from the cord this wide path is closed and all neurin generated in the afferent neurones passes directly through the reflex conduction-paths of the cord to the muscles, keeping the tone of both cord and muscles at a high level. It is, I think, highly probable that other instances of inhibition of nervous action may be explained along these lines.[1]

On awaking quietly after a night of deep sleep, I lie sluggish and heavy. I seem to be incapable of any muscular effort, in fact I can hardly close my fist, so slight is my voluntary power, and urgent appeals to my moral or intellectual nature may fail to bring me even to a sitting posture. If only gentle stimuli affect my sense organs, I gradually gather energy enough to enable me to crawl to my bath, and then, at the first splash of cold water on my skin, the slackness of muscle and of resolution disappears and I become again a normally active and alert individual. Any violent sensory stimulus, a very loud sound or a very bright light, will produce almost instantaneously a similar effect. During the night neurin has continually leaked away by the efferent nerves from the whole nervous system more rapidly than it is produced by the resting neurones, the potential of the whole nervous system has thereby been reduced to a very low level and evenly distributed throughout the system. A brisk stimulation of sensory nerves then quickly restores my voluntary power and general tone and alertness by causing a rapid production of neurin that diffuses itself throughout the nervous system. Again, if I stay in-doors all day taking no exercise, I grow restless and irritable; the generation of neurin has outpaced the discharge and the general level of potential has become too high. An hour's brisk exercise restores my normal equanimity; the vigorous efferent outflow reduces the general level of potential. Similarly a day spent in steady physical exercise leaves me in that condition of placid enjoyment of sitting still and doing nothing which is best known, perhaps, to mountain-climbers; the outflow has exceeded the generation of neurin, the general level of potential is low.

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[1] Prof. Verworn's paper, "Zur Physiologie der nervösen Hemmungserscheinungen" (*Arch. f. Anat. u. Phys.*, 1900), has come into my hands, through the kindness of the author, since the above was put into type. It is there shown that the inhibition of skeletal muscles is essentially passive inhibition, a cutting off of the stimulating influence proceeding from the motor centres. This fact emboldens me to suggest that in the reciprocal inhibitions of the motor centres of antagonistic muscles we have to do with a leading off of neurin from one path through the opening of a path of less resistance. The reciprocal inhibitions of visual images strongly suggest this view of inhibition.

What exactly is the nature of the process of discharge of neurin across the synapses and what is the part played by the inter-cellular substance can hardly be so much as conjectured, but we must assume that each discharge constitutes, or brings about in the inter-cellular substance, the delicate and specifically differentiated psycho-physical process. And we must further assume that each such discharge leaves the substance so changed that its resistance is permanently lowered. This permanent lowering of the threshold of the synapsis may be conceived to consist in any one of various changes, it may be that the thickness of the layer of inter-cellular substance between the terminals of the nerve-fibrils is diminished, or that its cross-section is increased, it may consist in some state of strain analogous to the strain of a dielectric substance remaining after discharge from a condenser, or in some molecular rearrangement, or more probably in some change of whose nature we cannot at the present time form any conception. This change, which seems to be the physical basis of memory and association, this organisation of the synapses, which corresponds to what Exner calls "Ausfahren" or "Ausschleifung der Bahnen," must be carefully distinguished from facilitation (Bahnung), which is a temporary condition due to the presence of a charge of neurin in the neurones. Exner himself observes the distinction, but by other writers the two things have been confused.[1]

It may seem to some a grave objection to the ideas propounded above, that they involve the assumption that the psycho-physical process underlying a continuous state of consciousness is a series of processes discontinuous in time. But such a purely *a priori* objection has no validity. It is conceivable that, if a creature whose vital tempo was set at a much higher rate than ours (as is perhaps the case in the gnat and other insects) were capable of being in some way directly affected by our psychical processes, then our psychical processes, thus objectively viewed, would appear to it as a series of pulses of consciousness separated by considerable intervals of time. We have seen that Richet does not scruple to assume that the nerve-wave, which he regards as the physical basis of consciousness or (if we may take his remarks literally) as identical with consciousness, is repeated only about ten times per second; and Herbert Spencer too teaches that our subjectively continuous states of consciousness consist, objectively regarded, in series of "pulses of feeling" corresponding to series of "nerve-pulses."[2]

My proposition, that the synapses are the seats of the psycho-physical processes, involves the corollary that the state of consciousness of an individual at any moment is determined by several, and, in any but the simplest states, by a very large number of psycho-physical processes occurring simultaneously in different parts of the cerebral cortex, the total state of consciousness being the resultant of purely psychical fusion of the several affections of consciousness determined at the several synapses. I am aware that this will not be readily accepted by many minds that have not realised that psychical fusion is a conception absolutely necessary for any but the most superficial consideration of the relation of physical and psychical processes. Psychologists, of course, make use of the terms "psychical com-pounds," "fusion," and "colligation"; but I believe that a large number of psychologists take the same view of fusion as Ebbinghaus, who writes,[1] "Das, was verschmilzt im Falle der Verschmelzung, sind mithin nicht die seelischen Gebilde, sondern die nervösen Prozesse." On the other hand, Helmholtz's theories of sensations of tone and colour involve the assumption of purely psychical fusion, and it is explicitly expressed in the following passage:—"Diese Thatsache ist wichtig, weil aus ihr hervorgeht dass der Inhalt jedes einzelnen Sehfeldes, ohnedurch organische Einrichtungen mit dem des anderen verschmolzen zu sein, zum Bewusstsein gelangt, und dass die Verschmelzung beider Sehfelder in ein gemeinsames Bild, wo sie vorkommt, also ein psychischer Act ist."[2] Exner lays down the principle of the multilocular excitation of a single state of consciousness perfectly clearly and explicitly when he writes:—"Eine Summe von Erregungen in der Gehirnrinde, verschiedene Bahnen betreffende, wirken als ein einheitliches Erregungsvorgang auf das Bewusstsein."[3] Sully[4] and Münsterberg[5] make statements similar to this. In my paper on vision I have used the phrase "psychical fusion" in the sense that is given to it by Helmholtz, and have pointed out that without this assumption it is impossible to explain many visual phenomena in terms of any of the rival theories.[6]

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[1] I made this mistake in the use of the term "Bahnung" in the essay to which I have several times referred. *Mind*, vol. vii.

[2] "Principles of Psychology," part ii, chap. i.

There is, I believe, no greater bar to the progress of our knowledge of mental processes than the frequently displayed reluctance fully to accept and to realise psychical fusion in this sense. This reluctance seems to be due to the deeply-rooted tendency to apply our conceptions of space to psychical processes, and to suppose that the soul can be acted upon only at one place at one time. It is this tendency that has led various authors to assume as the seat of the soul a "punktförmiges Seelenatom," a supreme monad, a pineal gland, or a central structureless parenchyma.[1] Of the many considerations that compel us to accept the assumption of a purely psychical fusion I will mention only one. If we do not accept it, we must assume that each state of consciousness is excited by a single complex psycho-physical process. Then, since no one state of consciousness endures more than a moment and no one ever recurs during the life of an individual, we shall have to assume as many million specific varieties of psycho-physical process as the moments of conscious existence experienced by any individual. This is an altogether impossible assumption; so immense a variety of states of consciousness can only result from the varying combinations of the affections of consciousness determined by a relatively few kinds of psycho-physical process. This view presents no difficulty so long as we do not lose sight of the fact that, as Rehmke[2] writes, "Die Seele hat, weil keine Grösse, auch keinen Ort: die Seele ist, aber sie ist nirgends"; the epigrammatic correctness of this statement is, however, a little too rigid, and the truth is perhaps better expressed by Lotze,[3] when he writes: "The soul stands in that direct interaction which has no gradation, not with the whole of the world, nor yet with the whole of the body, but with a limited number of elements; those elements, namely, which are assigned in the order of things as the most direct links of communication in the commerce of the soul with the rest of the world. On the other hand, there is nothing against the supposition that these elements, on account of other objects which they have to serve, are distributed in space; and that there are a number of separate points[4] in the brain which form so many seats of the soul. Each of these would be of equal value with the rest; at each of them the soul would be present, with equal completeness, but not therefore without any distinction; rather we might suppose that at each of them the soul exercises one of those diverse activities which ought never to have been compressed into the formless idea of merely a single outgoing force." [1]

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[1] Grundzuge der Psychologie," p. 573, Zweiter Halbband, Leipzig, 1902.
[2] *Physiog. Optik.*, S. 921.
[3] *Op. cit.*, S. 236.
[4] "Human Mind," p. 51.
[5] "Grundzuge der Psychologie," p. 556, Leipzig, 1900.
[6] *Mind*, vol. x., section v.

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[1] A suggestion made by Lotze in the *Medizinische Psychologie*, but afterwards rejected by him.

[2] "Die Seelenfrage," *Zeitschr. f. Psychol.*, *&c.*, Bd. ii.

[3] "Metaphysik," English translation, § 295.

[4] *i.e.*, synapses of the cortex.

Another difficulty, to my mind the most serious of all, must be frankly admitted. The synapses of the cord seem to differ from those of the cortex not in kind but in degree only; it might therefore be expected that, if the passage of the excitation-process across the synapses of the cortex constitutes or determines the psycho-physical process, then it should do so in the case of the synapses of the cord also. It may be noted, however, that a similar difficulty meets us even more decidedly if we assign the psycho-physical processes to the nerve-cells of the cortex or of any other part, for, in the case of the nerve-cells, we know of no important difference in structure or function between those of the cortex and those of the cord, whereas, as we have seen, we have strong reason to regard the synapses of these two regions as differing widely in the degree of resistance offered by them to the passage of the excitation-process. The difficulty may be met in either one of two ways. We may suppose that some obscure degree or kind of affection of consciousness is contributed by the synapses of the cord to that complex total resultant of many psycho-physical processes, coexistant in time but separated in space, which constitutes the state of consciousness of an individual at any moment. Or we may suppose that the discharge of neurin across the synapsis constitutes a psycho-physical process only when it takes place at more than a certain potential; as G. E. Muller writes: "But whether the brain-process can possess that peculiarity of being actually a psycho-physical process at any grade of intensity, remains wholly undecided."[1]

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[1] In his "Grundzuge der Psychologie" (p. 26), Professor Ebbinghaus asks, "haben wir sachliche Veranlassung, das Seelische in einen unbedingten und scharfen Gegensatz zu bringen zu der Art und Weise des Daseins, die wir als Ausgedehntsein und Materiellsein an Pflanzen, tierischen Leibern, u. s. w., alle kennen?" And he replies, "die Antwort ist nein, wir haben solche Veranlassung nicht." I believe that, on the contrary, the evidence for purely psychical fusion (of which evidence the facts brought together in this essay are an important part), enables us to answer this question with a very decided "Yes," and it seems to me that this evidence furnishes one, and perhaps the only, solid ground for regarding the soul as a being subject to its own peculiar laws, according to which the states or processes excited in it by the brain-processes, interact with one another, and so bring it about that the soul reacts upon the brain-processes to modify them. The same evidence suffices of itself, as it seems to me, to render untenable the doctrine of psycho-physical parallelism.

This paper may be fitly concluded by the quotation of an authoritative statement by Foster and Sherrington: "The higher psychical processes we may probably regard as being pre-eminently functions of the connexions of the several parts of the cortex with each other."[2] In the preceding pages I have merely attempted to expand and to define more fully the truth conveyed in this statement.

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[1] *Pyscho-physik d. Gesichtsempfindungen. Loc. cit.* p 4.
[2] "Text-book of Physiology," p. 1218.

A CASE OF COMPLETE ABSENCE OF THE VISUAL SYSTEM IN AN ADULT.[1]

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(Read before the College of Physicians of Philadelphia, November 6, 1901).

COMPLETE absence of the visual system is of very rare occurrence, especially in an adult. The study of the case I report in this paper should be of more value than one of agenesia of the visual system in a newborn child, because in the latter portions of the brain not belonging to the visual system are imperfectly developed, and faulty conclusions may therefore be drawn.

The boy, T. S., according to the case-books of the Pennsylvania Training School for Feeble-minded Children, was born in 1878, and was admitted to the School March 12, 1895. At the time of admission he weighed thirty-eight pounds, and his height was 3 ft. 7 in. He was an idiot, absolutely helpless, and very unclean. He could say "mamma," and nothing more. He was passionately fond of music. At the time of admission to the school he appeared to be about 8 or 10 years of age.

I saw him on July 30, 1897, and made a few notes on his condition. I regarded the case at that time as one of cerebral spastic paraplegia of the lower limbs, with absence of the eyeballs. The boy was unable to stand alone, and if he were stood up and not supported he fell backward. When supported he could take a few steps, but in doing so one lower limb was moved in front of the other (cross-legged progression). His knee-jerks were exaggerated, but ankle-clonus was not obtained. He could move his lower limbs while he was sitting, and there was not excessive rigidity of these limbs on passive motion. He uttered only a few monosyllables. His palpebral fissures were very small. He died March 2, 1900, and the necropsy was made two days later. The necropsical notes I shall use only in so far as they are of importance for the understanding of the case.

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[1] From the William Pepper Laboratory of Clinical Medicine (Phebe A. Hearst Foundation).

The body was that of a child apparently about 12 years of age (see fig. 1), though in reality his age was 22 years. No signs of puberty were present. The testicles were not in the scrotum; the right testicle, quite small, was found in the inguinal canal just above the pubes. The genitalia were like those of a child of 10 years. No hairs were found on the pubes, or in the axillæ, or on the face. The circumference of the head was 18¾ inches; the length of the body was 47 inches. The right upper limb could not be fully extended at the elbow, while the left upper limb could be fully extended. Slight contracture of the fingers of each hand was observed, so that the fingers could not be fully extended when the hands were fully extended at the wrists. The fingers could be fully extended when the hands were flexed at the wrists, as the contracture of the flexors of the fingers did not then interfere with the extension of the fingers. The lower limbs were slightly flexed at the knees, and could not be fully extended.

The palpebral fissure of each eye was very small, and the orbits contained only a small amount of what appeared to be fibrous connective tissue. Unfortunately, permission had not been given for the removal of the orbital contents. Nothing resembling an eyeball was seen.

The optic foramina did not exist, and it was impossible to find an opening in the usual situation of these foramina, and here there were merely slight depressions in the skull. No trace of optic nerves, chiasm, or optic tracts could be found (see fig. 2). There was no sign of an external geniculate body on either side, and the thalamus on each side had nothing resembling an optic tract passing from it. The posterior part of each thalamus was rounded and larger perhaps than one would expect to find it in a case of complete agenesia of the visual system (see fig. 3.) The brain was small, firm, and not œdematous. The left ascending frontal convolution in the centre for the upper limb was very small (see fig. 4), to which fact possibly was due the contracture of the right forearm (see fig. 1), because of imperfect development of the nerve fibres innervating this limb. The anterior colliculi of the corpora quadrigemina were fully as large and as well developed as the posterior. The occipital lobes were small, and the cuneus on each side (see fig. 5) was small, and the calcarine fissure short. The lower olives were covered by thick bands of nerve-fibres—*i.e.*, the external arcuate fibres were unusually well developed.

The spinal cord was small, but not otherwise abnormal.

The brain was hardened in formaline, and the basal ganglia of one hemisphere were cut in serial sections, while those of the other hemisphere were preserved intact as a gross specimen. Sections were made through the cortex of the left calcarine fissure, and stained by thionin and by Weigert's hæmatoxylin method. Frontal sections were made through the right occipital lobe in order that the optic radiations might be studied when cut transversely. The nerves supplying the ocular muscles were also cut and examined.

The cortex of the left calcarine fissure stained by thionin contained many nerve-cell bodies, but they were possibly slightly less numerous than are the cell bodies in sections from the corresponding region of a normal brain, especially in the third, fourth, and fifth layers. The giant-cells of the third layer were represented, but were possibly not as numerous as in a normal brain. The fibres of Vicq d'Azyr of the cortex of the calcarine fissure were not very distinct in sections stained by Weigert's hæmatoxylin method, but they were not entirely absent.

The optic radiations in the frontal sections of the occipital lobe were not entirely absent, but the area occupied by them was not very distinct.

Meynert's commissure was normal, and stained well by Weigert's hæmatoxylin method (see fig. 6).

Careful examination of the serial sections failed to show the slightest trace of an external geniculate body on either side, although the internal geniculate body was well developed. Fig. 6, a photograph from one of the sections, shows the internal geniculate body in its normal size, as well as the inferior posterior portion of the optic thalamus, but the external geniculate body, which should be present in a section at this level, is entirely absent. Comparison with fig. 326 of the *Anatomie des Centres Nerveux*, vol. i, by J. and A. Dejérine, is instructive. This drawing is from a section cut in the same direction as that represented in fig. 6 of my series, and shows the normal size of the external geniculate body and an optic tract in a section from this level. In the section of my series represented in fig. 6 there is no trace of an optic tract.

Some few medullated nerve-fibres are found within the pulvinar in my section, but those in the posterior portion of the thalamus are much fewer than those in the more anterior portions. The contrast is very striking, and is represented in a drawing of a horizontal section (see fig. 7), which should be compared with a corresponding section from a normal brain, as shown in fig. 321 of the *Anatomie des Centres Nerveux*, by J. and A. Dejérine.

The habenula in the horizontal sections appears well developed, and the fasciculus retroflexus of Meynert is well stained.

In the photograph the anterior colliculi of the corpora quadrigemina appear smaller than the posterior; but this is merely because the former were further from the camera.

Sections through one-half of the oculomotor nuclei stained with thionin contain many nerve-cell bodies belonging to these nuclei, and these cell bodies stain well with thionin and appear to be normal. They are possibly not as numerous as cell bodies in oculomotor nuclei of a normal brain. Sections through one-half of the oculomotor nuclei were also stained by the Weigert hæmatoxylin method.

Transverse sections of the extracerebral portion of the left oculomotor nerve near the cerebral peduncles stained with ammonium carmine show an apparently normal nerve. The nerve is possibly smaller than an oculomotor nerve from a normal brain, but it contains no sclerotic areas. It contains, however, some areas in which very fine nerve-fibres form distinct groups, but there is no scarcity of large nerve-fibres.

Transverse sections of the right oculomotor nerve are very similar to those of the left oculomotor nerve. The sections stain well by Weigert's hæmatoxylin method.

One of the trochlear nerves in transverse sections, stained either by ammonium carmine or Weigert's hæmatoxylin, appears to be normal, and contains nerve-fibres of good size. The nerve-fibres stain well with the Weigert hæmatoxylin, but in sections so stained the nerve-fibres are perhaps not quite so close together as in the trochlear nerve from a normal brain.

The nerve-fibres of the left abducent nerve stained in transverse sections by acid fuchsin are exceedingly small, much smaller than those of the oculomotor and trochlear nerves, but they stain well with Weigert's hæmatoxylin.

The right abducent nerve in transverse sections stained by acid fuchsin contains many nerve-fibres that are much larger than any of those found in the left abducent nerve, but it contains also many sclerotic areas. Some of the nerve-bundles stained by

Weigert's hæmatoxylin method contain very few medullated nerve-fibres. The contrast between the abducent nerves and the other two ocular nerves on each side is most striking (see fig. 8).

The nucleus of each abducent nerve contains many nerve-cell bodies which, with the thionin stain, appear to be normal.

The nerve-cell bodies of the anterior horns of the spinal cord in the cervical and lumbar regions stained with thionin are normal, but possibly a little smaller than those usually present in the anterior horns. No sclerotic areas are found in the lateral columns, neither are the nerve-fibres in these columns unusually fine in transverse sections.

von Monakow gives as primary optic centres the external geniculate body, the surface of the pulvinar, and the superficial gray matter of the anterior colliculi of the corpora quadrigemina. He says that Stilling, Bernheimer, and Kölliker believed that visual fibres arise also in the subthalamic body, the internal geniculate body, and the tuber cinereum. The anterior colliculi of the corpora quadrigemina in man, according to his view, have a very subordinate *rôle* in vision, and he believes that about 80 per cent. of the optic nerve-fibres terminate in the external geniculate body. My case of congenital absence of the central visual system confirms these views of von Monakow. It shows also that Meynert's commissure and the habenula are not part of the visual system. According to von Monakow (*Gehirnpathologi*) centrifugal fibres—*i.e.*, fibres having their origin in the visual cortex—are present in the optic radiations and pass to the inter-brain and mid-brain. The giant pyramidal cell bodies of the third layer in the cortex of the occipital lobe give off axones, which pass by way of the optic radiations and the arm of the anterior colliculus of the corpora quadrigemina, to terminate in the superficial medullary substance and gray matter about the aqueduct of Sylvius in the anterior colliculus. The Vicq d'Azyr fibres, he says, are often in a double row in the occipital lobe. They come, in greater part, from the fibres of the optic radiations which pass to the third layer of the cortex, but some of the Vicq d'Azyr fibres may belong to an association system. In long standing lesions of the optic radiations the Vicq d'Azyr fibres are very atrophic, and the double row of these fibres is scarcely recognisable. In my sections the comparatively few fibres passing to the pulvinar are doubtless the fibres described by von Monakow as arising in the occipital lobe, and in my sections the Vicq d'Azyr fibres are not very distinct.

von Leonowa has examined the microscopical sections from four cases of anophthalmia and three cases of atrophy of the eyeball, but in all these death occurred within a few months after birth. In all her cases the posterior colliculi of the corpora quadrigemina were intact, but the anterior colliculi were affected. von Leonowa[1] concludes from a study of the cases mentioned above that the posterior colliculi of the corpora quadrigemina have no relation to vision, while the anterior have; that the relation of the external geniculate body to the visual fibres is a very intimate one; and that the subthalamic body has no relation to vision. In none of her cases was the absence of the visual system complete, and all the brains were from children in the early periods of extra-uterine life.

In her cases the nuclei of the third, fourth, and sixth cranial nerves were intact, and this integrity is explained by von Leonowa as a consequence of the existence of ocular muscles. The external ocular muscles may be well developed, even though the eyeballs are exceedingly atrophic, as in a case of anophthalmia in a person of 27 years, reported by Haab, in whom the ocular muscles were normal in size and shape, although the ocular bulbs were very small. The nerves to the ocular muscles need not be absent in congenital defect of the visual system. In my case there was apparently no eyeball on either side, but in the small amount of fibrous tissue at the posterior portion of each orbit muscles may have existed, even though they had little or no function. One might suppose that in the case I have reported the third, fourth, and sixth nerves would have been very imperfectly developed in conformity with the law of Gudden, according to which atrophy in a neurone follows injury to this neurone. It should be remembered, however, that in my case the nerves supplying the ocular muscles were not injured, and may have been in union with ocular muscles, as in Haab's case.

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[1] *Archiv. für Psychiatrie*, 1896, vol. xxvii, p 58.

It has been shown by von Leonowa,[1] in the report of a much-quoted case, that nerve-fibres may develop, although they apparently have no function. She examined a fœtus in which the brain and spinal cord had not developed, and yet the vertebral canal was filled in its entire extent by posterior nerve-roots. Some of the fibres of the posterior roots extended from the lower part of the canal to the upper, as they would have done if they had formed the posterior columns of a normal spinal cord. All the posterior roots ended blindly, and seemed to have sought a spinal cord in which to bury themselves. No anterior roots were found. All the peripheral nerve-fibres present had their origin in the spinal ganglia, and belonged, therefore, to the sensory system. The muscles of the limbs appeared fully normal, although no motor nerves existed. Most of the axis cylinders of the posterior roots were non-medullated or had only a thin medullary sheath. The cell bodies of the spinal ganglia were as numerous and as well developed as in a normal fœtus. The absence of medullary sheaths on the posterior roots, von Leonowa thinks may have been caused by the amniotic fluid, inasmuch as medullary sheaths were present on axones that were covered by the skin.

Gade[2] and Karl and Gustaf Petrén have found posterior roots growing into the spinal canal in complete failure of development of the spinal cord. I am unable to determine from the statements of the latter authors whether in their case or in Gade's the individual fibres of the posterior roots were normal or not.

With the results of these investigations in mind we can understand that nerves might grow equally well into the orbits and persist, even though the ocular muscles were absent. Indeed, von Leonowa and Karl and Gustaf Petrén have shown also that in early life muscles may be developed, although no motor nerves exist, so that the converse may well be true—*i.e.*, motor nerves probably may exist, although the muscles are not developed. All the ocular nerves in my case, however, were not normal. The two sixth nerves were very imperfectly formed.

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[1] *Neurologisches Centralblatt*, 1893.

[2] Gade, cited by Karl and Gustaf Petrén. *Virchow's Archiv.*, 1898, B. 151

Meynert's fibres are asserted by some to be the connection between the optic nerves and the oculomotor centres, although such distinguished cerebral anatomists as Edinger and von Monakow tell us that the anatomical proof for this assertion is wanting. Recent investigations seem to indicate that some of *these* Meynert fibres (for there are more than one band of fibres named after Meynert) belong to a bundle extending into the anterior columns of the spinal cord. In my case the Meynert fibres in the corpora quadrigemina were well developed, although there could have been no reflex fibres from the iris to the oculomotor centres, so that we must conclude that the function of these Meynert fibres cannot be merely to serve the pupillary reflex. I am unable to give the course of the so-called pupillary fibres from a study of my preparations, as the fibres in the region of the corpora quadrigemina form an intricate network.

It is not surprising that cell bodies should be numerous in the cortex of the calcarine fissure in a case of congenital absence of the visual system. Some of these cell bodies have axones which are situated in the optic radiations and terminate in the primary optic centres—*i.e.*, the external geniculate body, the pulvinar of the thalamus, and to a less extent the anterior colliculi of the quadrigeminal bodies, and these fibres probably are not directly concerned in vision. The visual cortex is intimately connected with widely separated portions of the brain, as shown both by physiological and anatomical investigations. The movements of the limbs, the functions of speech, and especially the movements of the eyeballs, are in intimate relation with vision, and important association tracts between the visual area and other parts of the brain can be demonstrated anatomically. The cell bodies of the visual cortex must have axones in relation with remote parts of the cerebral cortex, and, therefore, it is not surprising that these association neurones are developed even in a case of congenital defect of the visual system. von Monakow represents in his *Gehirnpathologie* (p. 265) a section of a calcarine cortex from a case of lesion of the optic radiations. The giant pyramidal cell bodies of the third layer of the cortex had entirely disappeared, and the third, fourth, and fifth layers were degenerated in a way they were not in my case. It should be remembered that in von Monakow's case the axones destroyed were those of cell bodies situated in the calcarine cortex, and that atrophy of these cell bodies probably followed the injury of their axones. This did not occur in my case, and probably the nerve-fibres present in my specimens in the optic radiations arose in the occipital lobe. It is difficult to understand what the function of these fibres can be.

Although the individual described in this paper was an idiot, we should be cautious in attributing his deficient mental development to the absence of his visual system. It would seem probable, however, that the entire brain was in a state of imperfect development.

The spastic paraplegia of the lower limbs deserves mention. The nerve-cell bodies of the anterior horns in the lumbar cord were normal by the thionin stain, except that they were not so large as the cell bodies of this region usually are, and the lateral columns did not contain sclerotic areas. This is not the first time I have found an apparently normal spinal cord in congenital spastic paraplegia, and I think the explanation is to be found in an imperfect development of the motor neurones. A deficiency from birth in the number of these neurones would probably have the same result as partial destruction of the central motor tracts, and in the absence of the development of motor neurones there would be no sclerosis, inasmuch as no space existed to be filled by proliferation of the neuroglia. Indeed, sclerosis may be entirely absent if destruction of nerve fibres occurs in early life, because the growing tissue of the spinal cord fills the space left by degenerated fibres, and proliferation of the neuroglia does not occur. I have seen the spinal cord in a case of infantile hemiplegia presenting little or no sclerosis, although the person from whom this cord was removed had lived many years after the paralysis had developed. The case to which I refer was in Dejérine's service. In two cases of congenital spastic paraplegia of the lower limbs I believed I was able to detect an unusual fineness of the nerve-fibres of the pyramidal tracts, but I was unable to do so in this case, T. S.

I am indebted to Dr. de Schweinitz for the reference to a case reported by W. S. Little[1] in 1885, in which congenital absence of some portions of the visual system was found. The brain was from a child of ten years of age, who had been an inmate of the Pennsylvania Training School for Feeble-minded Children, and the specimen was examined by A. W. Wilmarth. This specimen and mine have therefore come from the same institution. The child had had bi-temporal hemianopsia. There was no optic commissure, but a small projection was found on the inner side of each nerve where the commissure should have been. These projections did not touch by at least one-quarter of an inch. A complete study of the specimen does not seem to have been made.

A still more important case, and one more closely resembling mine, has been reported by Haab.[2] His specimen was from a girl, twenty-seven years old, who had the development of a child of fourteen or fifteen years, and from her appearance had not reached puberty. She was an idiot, and had much difficulty in walking. The optic chiasm was entirely absent, but the optic tracts were present as small bands. The oculomotorius, abducens, and trochlearis appeared to be normal, and yet the merest trace of an eyeball was found. The muscles of the orbits appeared to be normal. The external geniculate body was entirely absent, but the internal geniculate body was of normal size. The corpora quadrigemina were of normal size macroscopically. The optic foramen was almost obliterated. The pulvinar of the optic thalamus was smaller than in a normal brain.

The conclusions that may be drawn from my case are:

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[1] *Transactions of the American Ophthalmological Society*, 1885, p. 367.

[2] *Beitrage zur Ophtalmologie als Festgabe.* Friedrich Horner, 1881, p. 131.

(1) The chief "primary" optic centre is the external geniculate body.

(2) The pulvinar of the optic thalamus is also an important "primary" optic centre.

(3) The anterior colliculus of the quadrigeminal body in man has an unimportant relation to vision.

(4) The subthalamic body, the habenula, the internal geniculate body, probably are not part of the visual system.

(5) The cortex of the calcarine fissure may contain nearly the normal number of cell bodies, even though the visual system may be undeveloped.

(6) The nerves to the ocular muscles and their nuclei may be developed, even though the visual system is absent.

(7) Congenital spastic paraplegia may be the result of deficient formation as regards number or size of the neurones of the central motor system, even though such a deficiency may be difficult to detect by the microscope.

FIG. 1.

A photograph of T. S., taken after death. The right upper limb, the fingers of both hands, and the lower limbs are slightly contractured. The palpebral fissures are small.

FIG. 2.

The occipital lobes have been cut away. A portion of the right temporal lobe has also been cut away in order to show the complete absence of the visual system. The internal geniculate body (A) is well shown, but no trace of the external geniculate body or of an optic tract or chiasm can be found.

FIG. 3.

The optic thalami are well shown. The anterior colliculi of the corpora quadrigemina, especially the right colliculus, appear smaller than the posterior, because the former were further from the camera.

FIG. 4.

The left ascending frontal convolution in the centre for the upper limb (B) is very narrow.

FIG. 5.

The upper part of the right cerebral hemisphere, showing the small cuneus (C).

Fig. 6.

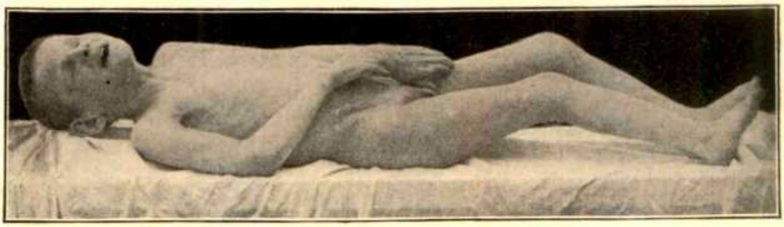
Photograph of a horizontal section of one cerebral hemisphere, showing Meynert's commissure (B) well developed, and also the internal geniculate body (C), while the external geniculate body is entirely absent

Fig. 7.

A drawing of a horizontal section through the basal ganglia of one hemisphere. The fibres entering the posterior part of the optic thalamus are few in number.

Fig. 8.

Drawings of transverse sections through portions of the abducent nerves. The upper drawing represents the fine fibres of the left abducent nerve, and the lower drawing the sclerotic areas in the right abducent nerve.



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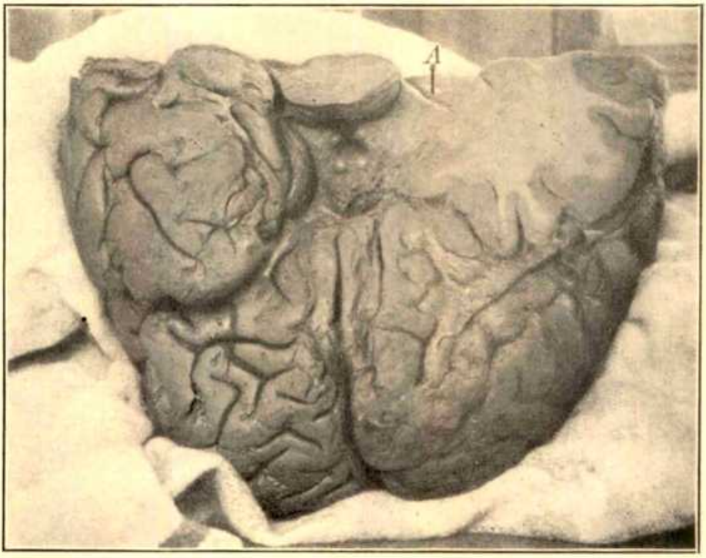
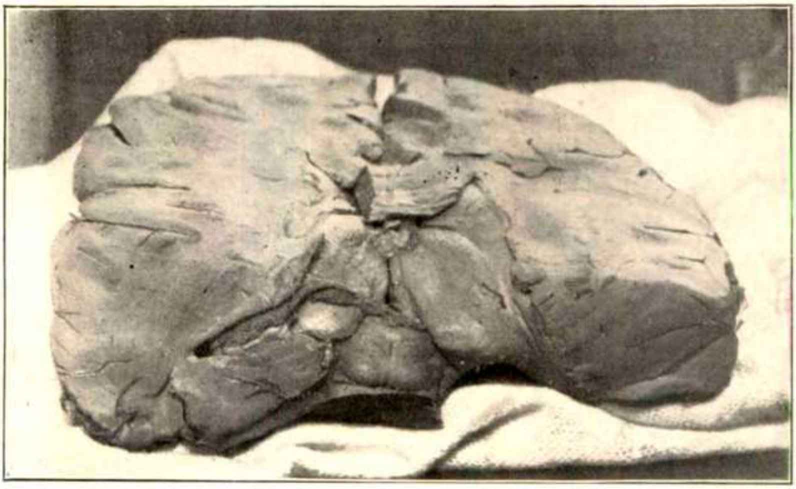


FIG. 2.



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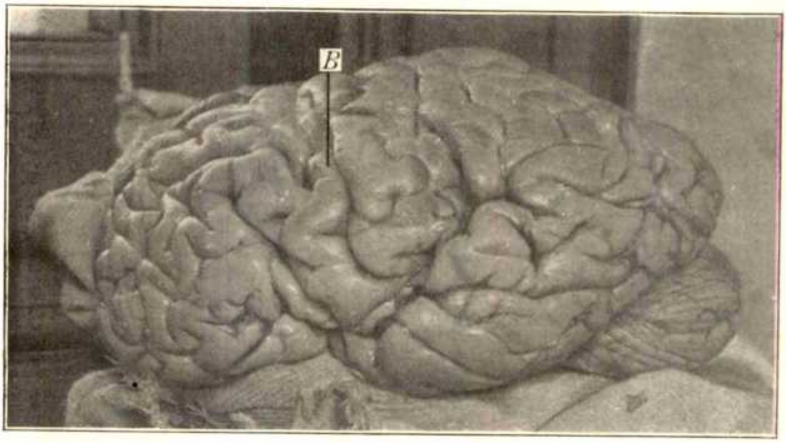

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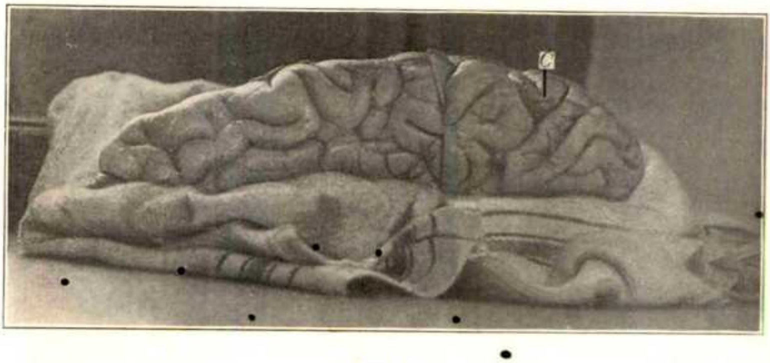

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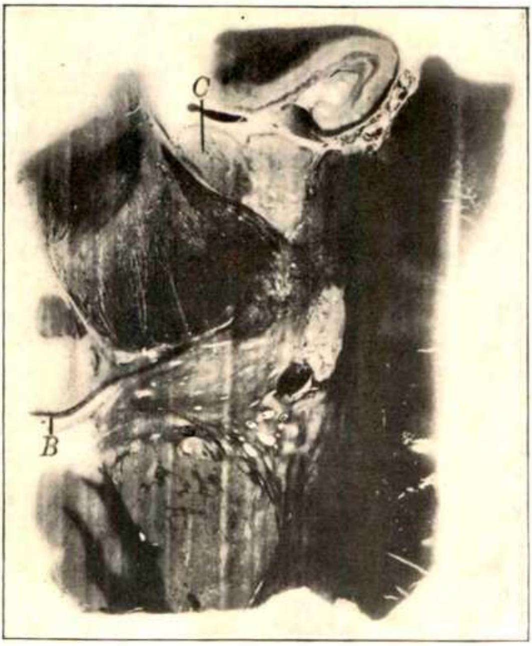

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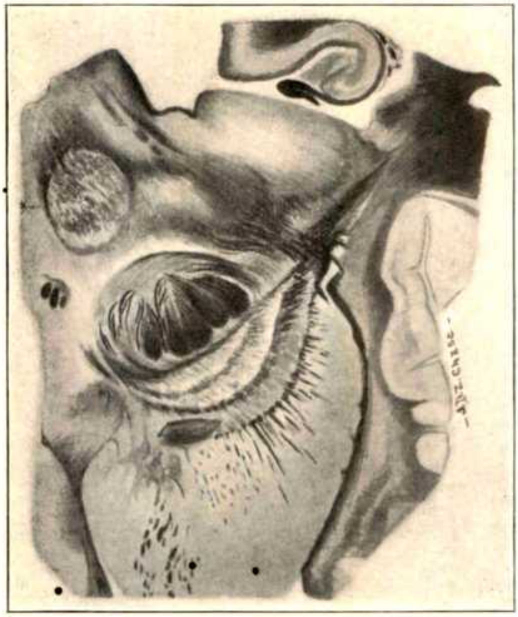

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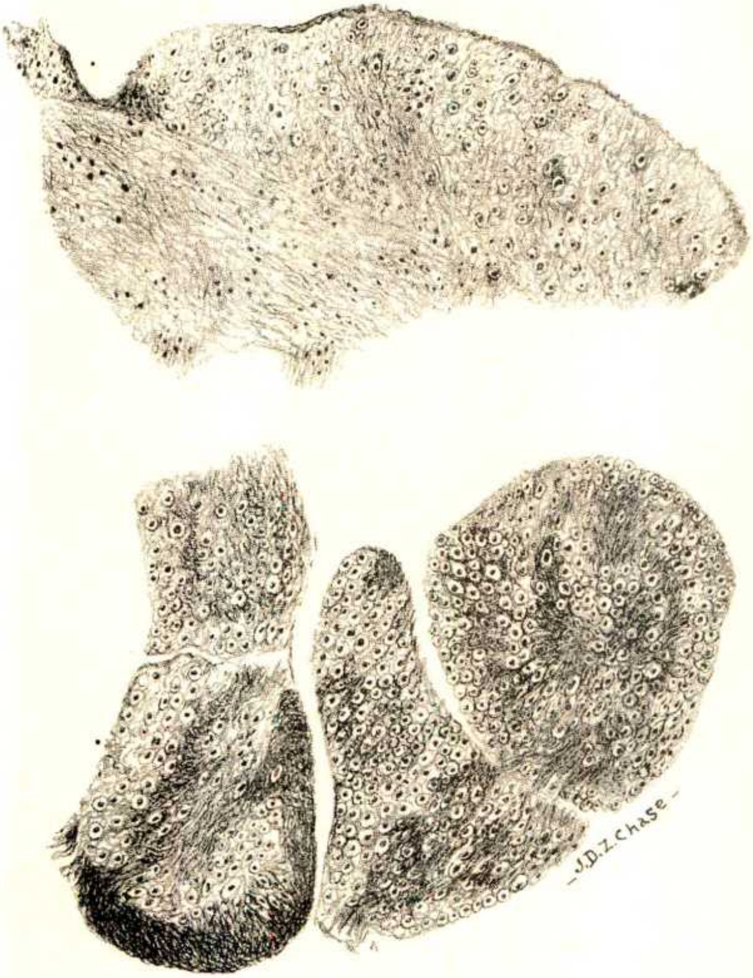

FIG. 8.

THE AREAS OF CUTANEOUS DISTRIBUTION OF THE 1ST SACRAL AND 5TH LUMBAR NERVES AS ILLUSTRATED BY TWO CASES OF HERPES ZOSTER.[1]

BY T. ALDOUS CLINCH, M.D.
Nottingham.

CASE 1.—*In the Area of the First Sacral Nerve.*

THE extreme rarity of eruptions of herpes zoster in this area invests the following case with more than common interest. Head has described but one in all his great experience, and that associated with simultaneous affection of the fifth lumbar area.

I am not acquainted with any other case, but my opportunities of studying the literature have been very limited.

The patient I attended for Dr. Collins, Nottingham, to whose generosity I am indebted for permission to publish the following account.

History.—Patient was a domestic servant of rather low type, about 50 years of age, and probably syphilitic. On August 29 the right great toe became numb; on the 30th it was red and painful, and on the 31st black. At this date the eruption appeared: a small circular patch about the size of a florin, slightly external to the posterior superior spine of the ilium; a second just posterior to and immediately below the great trochanter, about one and a half long by one inch broad; a third below and slightly posterior to the head of the fibula. All these patches were of moderate severity, and not preceded by the sensations and signs shown in the foot.

Present state.—When I first saw her, five days after the onset of the attack, an area of the foot—comprising the outer half of the sole, the tread of the foot, and the terminal phalanx of the great toe on both plantar and dorsal aspects—was reddish black; it ached and was painful and hot to the touch. Through the thickened epithelium appeared a few dimly seen yellowish spots. I was shown no more at that visit, and my hasty diagnosis was a diffuse superficial cellulitis. I am sure a correct diagnosis was possible, and I hope I could make it another time.

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[1] This paper was read before the Nottingham Medico-Chirurgical Society, December 4, 1904.

Case 1.

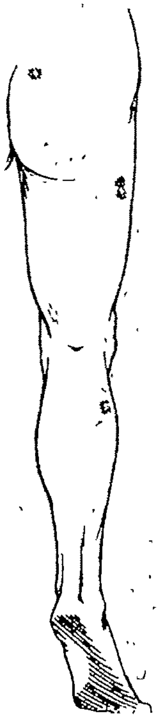


Fig. 1.
Area of distribution of herpes in Case 1.

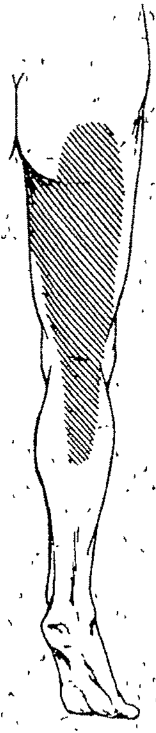


Fig. 2.
Area of distribution of small sciatic (copy from Quain).

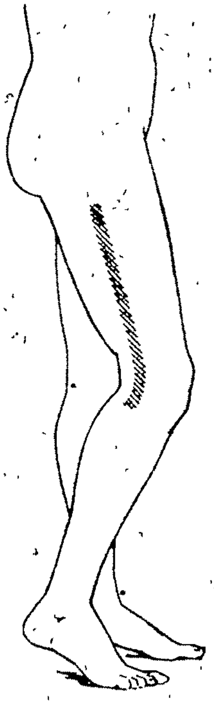


Fig. 3.
Probable area of distribution of first sacral on thigh.

Treatment ordered was poultices with recourse to the "knife" later, if necessary.

Progress.—The next morning the other areas were displayed to me and a mistaken diagnosis was no longer possible. The poultices had afforded much relief to the painful symptoms in the foot.

Constitutional malaise, which was very severe for the first few days, had also somewhat subsided. Future progress presented no conditions worthy of further attention.

'I very much regret not having made a complete examination of the nervous system, but its importance did not occur to me till too late.

Remarks.—The further and more exact anatomical diagnosis of the primary lesion appeared at first sight difficult, but I hope that my reasons for locating it as I have indicated in the title will carry conviction to others besides myself.

The first point that struck me was that the foot lesion corresponded almost exactly to the area allotted by Head to the first sacral; but above and beyond this area were three areas which did not coincide with any one known area. Dr. Head has laid it down that there are three maximum points of attack in a case of herpes: (1) that corresponding to the posterior primary division; (2) that corresponding to the lateral branch, and (3) that corresponding to the anterior branch of the anterior primary division of the nerve.

The sacral patch corresponds fairly closely with the area supplied by the posterior primary division of the first sacral, as displayed by the method of dissection, and it is, in my opinion, not assuming too much to consider that this patch is due to lesion of the first sacral. It moreover lies immediately above an area allotted by Head to the third sacral.

In normal cases the small sciatic nerve-trunk draws its supply of fibres from the second, third, and first sacral nerves, the order given being that of the predominence of the fibres. Now the area of skin supplied by the small sciatic, as shown in the last edition of Quain, corresponds very closely with that given by Head as the combined area supplied by the second and third sacral. Head, however, gives no area in this locality for the first sacral, which obviously must have some skin to supply, seeing that some fibres from it enter the small sciatic trunk. The second sacral being arranged pre-axially to the third sacral, it is not illogical to assume that this first sacral area, *which must* *exist*, will be on the pre-axial border of the second sacral area. Now it is on this border that we find the two remaining patches of herpes in this case, and I believe that they indicate the existence of a narrow strip of skin along the pre-axial border of the second sacral area, which is supplied by the first sacral, that they may indicate its extent upwards and downwards, and that it is probable that this area is very narrow in its mid portion, and may be actually separated in two.

If my hypotheses are correct the first sacral supplies an area of skin corresponding to its posterior primary division, close to the posterior superior iliac spine, a narrow strip of skin extending from the great trochanter to below the head of the fibula, corresponding to the lateral branch of the anterior primary division, in addition to the area on the foot and leg already described and figured by Head.

For my sketches I am indebted to Dr. Ferraby, to whom and Dr. Collins I have to tender my warmest thanks.

CASE 2.—*Occurring in the Area of the Fifth Lumbar.*

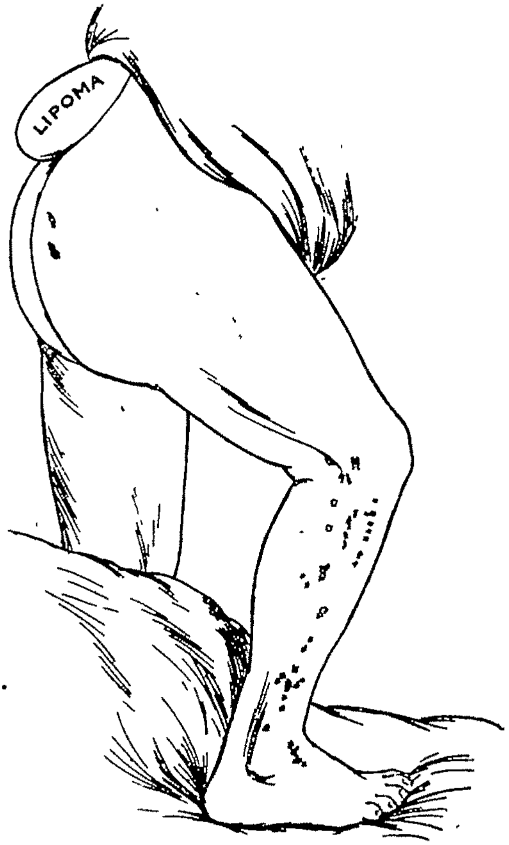
I am indebted to the kindness of Dr. Ferraby, of the Branch Dispensary, Nottingham, for this case. Dr. Ferraby, in addition to allowing me to report the case, has been so good as to draw a most graphic sketch of the parts affected.

It is of special interest on one point, and is also sufficiently rare to be worthy of record. Head has reported two uncomplicated cases and one complicated with simultaneous affection of the first sacral.

The details briefly are as follows:—

The patient, an old woman of 70, on Thursday, October 17, experienced the premonitory symptoms of the disease over an area on the buttock, some inches from the cleft, and on a level with its commencement. A week later the rash appeared: two small areas of vesicles separated by an interval of about one inch, and simultaneously pains commenced in the leg area, which corresponds so closely with that given by Head that it needs little further description. The rash—which, according to her account, came out a week after the buttock area—perhaps hardly descended so low on the foot, while it ascended to the level of the outer condyle of the femur, a somewhat higher level than that given in Head's diagrams, belonging, therefore, to class called by him "prefixed."

I obtained the foregoing history a week later still, when I saw her for the only time, and on that occasion she still had some pain and neuralgia, but the vesicles had dried and the scabs had been removed for the most part. There was no change in sensation to touch, pain, heat and cold, as compared with the other limb. The methods used were somewhat coarse. The knee-jerk was markedly impaired, that of the other limbs being, I believe, increased to some slight degree.



CASE 2.

This cannot be attributed to interference with fibres connected with the path of the reflex arc; for though the knee-jerk is not a true reflex, yet the reflex arc must be complete in order that it may be present.

The interference with the fifth root must be supposed to have lowered the tonus on the same side of the spinal cord, while it may have possibly slightly irritated the opposite side, thus illustrating the law that stimulation, irritation and depression are effects resulting from the same cause acting with different degrees of intensity. I could detect no abnormality in the plantar reflex.

The point perhaps of most interest is the buttock area; as in the first case it has not been previously described. I regard it as indicating the area supplied by the posterior primary division, and it further helps one to see that on the buttock, which up to now has seemed such a medley of areas, there is the same order and systematic arrangement as in the other parts. Dr. Head tells us that many of these lower areas have been hardly more than guessed at.

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CLIFFORD ALLBUTT. "System of Medicine." Article by Dr. Head.
BRAIN, vol. xxiii., No. 91. "Pathology of Herpes Zoster, &c." Drs. Henry Head and A. W. Campbell.

TWO CASES OF ASTEREOGNOSIS.

BY THEODORE DILLER, M.D.

Pittsburg, U.S.A.

As a contribution to the study of the perversion of the stereognostic sense I desire to place on record the two following cases :—

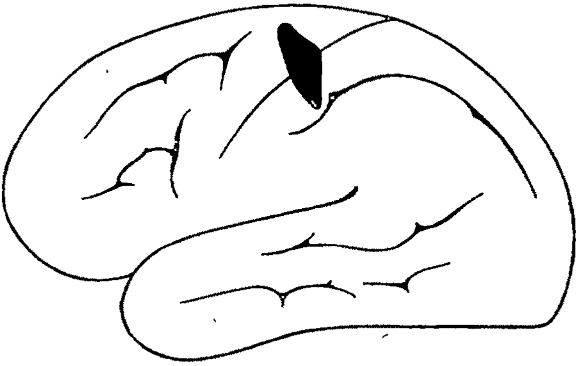
Case 1.—A single man, aged 40, was nineteen years ago struck in the left parietal region by a brick, which badly fractured the skull, necessitating the removal of a considerable piece of bone. He immediately lost consciousness, and he has no memory for the four months succeeding the accident.

He sustained a left-hemiplegia, which disappeared five months after the accident (one month after he "came to"). But a certain "clumsiness" of the right hand remained and has persisted up to the present time, so that while the hand became as strong as before the accident, its usefulness was, in a large degree, lost. A few months after the accident he was seized with an attack of typhoid fever, from which he made an excellent recovery. One year after the accident he contracted a chancre, which was followed by secondary symptoms.

For six or seven years past he has had difficulty in passing his urine, being compelled to make extra muscular efforts to accomplish the act and only partially succeeding in emptying the bladder. The stream is small and often interrupted. For the past two or three years his sexual powers have been declining, and during this period his vision has been steadily failing. Two years ago his left eye "turned out" rather suddenly, but after several weeks it returned to the normal position. For eighteen months past he has staggered in walking, which circumstance gave rise to the suspicion that he was drunk.

Examinations were made on September 6 and several subsequent dates. In the left parietal region there is a deep depression in the skull about 3 inches long, tapering at both ends, and about 1 inch wide in the middle just over the middle of the central convolutions, extending from 1 inch to the left of the median line, downward and slightly backward, crossing the line of the Rolandic fissure about its middle like the letter X (see figures).

The knee-jerks are absent. There is a moderate degree of static and locomotor ataxia. Eyes:—Pupils: right, 3½ mm., left, 3 mm. in diameter. Neither react to light, but both react slightly to accommodation. Vision is very dim. In both eyes advanced optic atrophy was discovered (Dr. Curry). When examined, stripped, no loss of motion or atrophy can be discovered. He recognises promptly in all parts of his body,



A rough diagram intended to illustrate the relationship of the depression in the skull to the central convolutions in Case 1.

Photograph of patient in Case 1, with a piece of white paper placed over the skull depression, to indicate roughly its area and position.

contact, pain, heat and cold. He is not able to recognise objects placed in his right hand, *e.g.*, coins, corks, watch, bottle, pocket-book, key, ring, card, pocket-knife and handkerchief. When these objects are placed in the left hand he recognises them instantly. On the palm of the right hand he recognises the two points of the æsthesiometer as such only when these are at least 60 mm. apart on the fingers and 80 mm. apart on the palm, and at still greater distance apart on the arm and back of the hand and on forearm. But with the right hand he tells many of the qualities of objects placed within it, *e.g*,, hard, soft, smooth, rough, wet, dry, heavy, light, warm or cold. Indeed, he estimates small differences in weight and temperature correctly. Even with the hand lying prone upon a table he estimates accurately small differences in weight (*e.g.*, dime and quarter). With either foot he recognises the length or point of a pencil.

When the fingers and wrist of the right hand are passively placed in different positions of flexion, extension, ab- and adduction, he is unable to reproduce these positions in the other hand. But he promptly reproduces such positions in the right hand when made in a similar manner in the left. Positions of the shoulders and elbows are reproduced correctly on either side.

REMARKS.—That the case is one of tabes, that the astereognosis was produced by the head injury, and that the astereognosis is not a symptom of the tabes, all seem clear. The extensive skull depression seems to be filled in with fibrous tissue. The head injury must have been very severe indeed and the general commotion extensive. That the central convolutions, in their middle third, were the brain structures which were most severely involved by the traumatism is borne witness to by two facts: (1) the history of hemiplegia following the injury, and (2) the location of the skull depression. That the pathological processes induced by the injury were largely dissolved is borne witness to by the fact that after hemiplegia had existed five months it then disappeared. But that certain pathological changes of a permanent character remain seems clear when the long persistence of the astereognosis (over eighteen and a half years) after the disappearance of the hemiplegia is considered. Where are these changes which are producing the astereognosis in this patient? While this question cannot, of course, be answered positively, yet the probabilities point, with very considerable force, to the middle region of the central convolutions as the chief seat of these changes. But how widespread they may be can only be conjectured.

In brief, the evidence in this case, so far as it goes, points to the middle third of the central convolutions as the cortical location of the astereognosis sense or judgment, and that cells other than the motor cells of this region are concerned in the appreciation of this sense. The case also affords evidence in favour of the view that all sensory impressions are received in cortical regions other than the central convolutions (pain, contact, temperature senses intact).

If the theory of Walton and Paul[1] be adopted as a working hypothesis, this case may be explained upon it. According to this hypothesis the "lower grade" of sensory memories (contact, pain, temperature) for the hand and arm are received in the cortical area posterior to the Rolandic convolutions, *i.e.*, in the parietal or post-Rolandic region, while in the middle portion of the central convolutions the various sensory impressions coming from the arm to the post-Rolandic area are assembled to form the stereognostic sense or judgment.

Dejérine[2] says that it never occurs that all forms of sensation, superficial and deep, are perceived when astereognosis is present, *i.e.*, that astereognosis alone is never seen. Certainly in both the cases here presented the reception of one sort of so-called sensory impulse was strikingly defective, viz., that of the spacing sense, while another, the position sense, was somewhat altered, although not absent. Dercum[3] found spacing sense almost uniformly absent in a large number of cases of astereognosis which he studied, and concludes that loss or impairment of the spacing sense is the most important factor in determining astereognosis. He also concludes that "next in importance to the spacing sense appears to be the knowledge of the position of the fingers and ataxia of movement." Commonly "spacing sense" and "position sense" are absent (or defective) when astereognosis is present; while the ability to perceive the "lower grade" of sensory impressions (pain, contact, temperature) is present. This is true of both my cases. But neither the "spacing" or "position" senses, it seems to me, are simple sensory impressions, as, *e.g.*, pain, temperature, contact, *but rather expressions of stereognosis*, and that their loss is due to the inability to properly assemble and coordinate sensory impressions received, and not factors in producing it. *In other words, "spacing" and "position" senses are component elements of the stereognosis judgment.* If this be allowed it could safely be said that all forms of primary sensory perceptions are present in the case related, unless, perhaps, muscular sense, and we cannot be sure this is absent.

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[1] BRAIN, Autumn, 1901, pp. 432, 433.
[2] *Semiologie du Système Nerveux*, p 890.
[3] *Jr. Nerv. and Ment Diseases*, 1900, p. 569, *et seq.*

Case 2.—A metal polisher, aged 35, has been married eleven years. Fifteen years ago he contracted syphilis. For many years he has used tobacco and alcohol to excess.

In June, 1900, he was, without warning, seized with a general convulsion of three hours' duration accompanied by loss of consciousness. A month later a second general convulsion occurred, which lasted five hours. A sensation of dizziness and "darkness before the eyes" gave him five minutes' warning of the approach of this attack. In October following he had a third similar convulsion of fifteen minutes' duration, ushered in by the same aura. Last November he was struck, while in a brawl, a severe blow on the head. . Ever since this time he has had "numbness" in the right face, arm and leg. In January he began to suffer from severe and persistent headaches, which continued up to August, since which time he has been free from them. This headache, at times, was so severe that he was compelled to go to bed. Vomiting was not a symptom.

Examination on April 26 discovered optic neuritis in the right eye (Dr. Curry). The left nerve could not be examined because of a corneal opacity resulting from an old injury. Stripped, he exhibited no muscular atrophy. Dynamometer: right, 83; left, 76. The knee-jerks were both present, the right more prompt than the left, but scarcely exaggerated. The Babinski toe reflex was absent. Station and gait seemed normal.

Sensation.—From all parts of his body sensations of contact, pain and temperature are normally perceived. As a rule, he is unable to recognise various objects placed in his right hand. He was tested with various coins, paper money, pencil, bottle, ring, watch, cards, cork, pocket-knife, key, &c. He could tell very much concerning objects without being able to say what they were. He appreciated even slight degrees of heat and cold and small differences in weight. He could tell whether objects were large or small, smooth or rough, hard or soft, wet or dry. He correctly recognised a pocket handkerchief and knew that a card was pliable, and suspected it might be a piece of leather. All these objects, when transferred to the left hand, were quickly recognised.

When the right hand and fingers were placed in various positions of flexion, extension, ab- and adduction, these positions could usually be imitated by the other hand, but not always and never without hesitation and apparent conscious effort. This was in strong contrast to the prompt and invariably correct reproductions in the right hand and fingers, when the left were in a similar fashion passively moved. The ability to recognise two points as such ("spacing sense") showed that they could be so recognised only when 40 to 60 mm. apart on the palmar surface of the fingers; and when 60 to 80 mm. apart on the palm itself. On the right arm and forearm two points could be recognised as such when 30 mm. apart in the transverse direction, but only when 80 to 125 mm. apart in the longitudinal direction.

On October 29 last the optic neuritis had subsided, leaving an atrophy behind (Dr. Curry). The urine was found to be normal.

Remarks.—A consideration of the convulsions, severe headaches, optic neuritis going on to atrophy, taken together, clearly point to a gross intracranial lesion; and the probabilities, I believe, favour a tumour rather than a diffuse syphilitic process. The convulsions cannot be considered as giving any hint as to the location of the lesion, since they were always general in character. Can the astereognosis be considered as a localising symptom? It may at least, I believe, be so regarded in that it indicates that the disease process is situated in the left cerebral hemisphere. Does it indicate anything more? I believe that when the absence of cranial nerve involvement in the case is considered in connection with the accumulated evidence favouring the central or parietal convolutions (or both) as the seat of the stereognostic sense it would be reasonably safe to diagnose a lesion of the cortex, involving either the Rolandic or parietal region or both these areas of the cortex; and with the evidence afforded by my first case and from a consideration of the arguments adduced by Walton and Paul[1] I should favour trephining over the region of the middle third of the Rolandic region, in case of operation, rather than over the post-parietal convolutions, in which region it is held by Mills[2] impressions of cutaneous sensation and muscular sense are perceived, and where stereognostic judgment takes place. Certainly the first case here reported argues strongly against Mills' view that the cortical centres for stereognosis are entirely separate from the motor senses proper (see diagram).

An operation may yet be performed on this patient, a study of whose case affords an example of the extreme desirability of the ability to localise the stereognosis sense, considered from the purely practical point of view alone.

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[1] *Loc. cit.*

[2] "Text-book on Nerv. and Ment. Dis.," p. 341 and p. 257 *et seq.*; also *Journal Nerv. and Ment. Dis.*, November, 1901, p. 599 *et seq.*

Reviews.

Vorlesungen über die pathologische Anatomie des Rückenmarks. Unter Mitwirkung von Dr. SIEGFRIED SACKI, Nervenartzt in München, herausgegeben von Dr. HANS SCHMAUS, A. O. Professor und 1. Assistent am patholog. Institut in München. (J. F. Bergmann, Wiesbaden, 1901.)

THIS book, which has already attained a wide popularity in Germany, is based upon a course of lectures delivered by Professor Schmaus upon the pathological anatomy of the spinal cord. With the collaboration of Dr. Sacki the original material of those lectures has been amplified and extended so as to present a very complete account of the present-day views on the subject.

The various diseases of the cord are approached mainly from the anatomical standpoint, clinical considerations being but briefly referred to. Yet the clinician who studies this work cannot fail to gain much useful information.

Of the twenty-one lectures which constitute the book, the first four are devoted to the general anatomy of the cord and its various elements. The chief spinal tracts are enumerated, and the origin, extent and termination of each is discussed. With regard to the neurone theory, although the authors do not consider its position altogether unassailable in the light of recent researches, yet it affords a useful basis on which to study system degenerations.

The structure of the various types of nerve-cells is clearly described and illustrated, together with the changes which they undergo under the influence of fatigue, fevers, organic and inorganic poisons, &c. The term "chromatin," as applied to the Nissl granules of the nerve-cells, is deprecated, and Lenhossek's term of "tigroid substance" is adopted instead—the former name belonging strictly to the nuclear chromatin, an entirely different substance. Tigrolysis is a condition the result of reaction of the nerve-cells to various influences, not a primarily degenerative process. It may or may not be accompanied by disturbance of function.

Wallerian or secondary degeneration of nerve-fibres, the result of interruption between the axis-cylinder process and the nerve-cell or of disease of the cell, is described and contrasted with primary degeneration of nerve-fibres, which occurs in the absence of both of those conditions. Many, if not all, of the primary degenerations are regarded as due to the influence of toxins, and occur in various systems of fibres, in single tracts, or in groups of tracts habitually co-ordinated in function. Examples of such primary toxic system diseases are afforded by ergotism, lathyrism, chronic alcoholism, lead and other metallic poisonings, blood diseases, auto-intoxications, and cachectic diseases.

Three lectures are devoted to the pathological anatomy of tabes dorsalis. After a description of the spinal degenerations, essentially of exogenous tracts, in this disease, the elective distribution of the various degenerations is discussed. The posterior root-fibres do not degenerate equally or simultaneously; some are affected earlier than others. Some observers account for this by the various functions of these fibres, whilst others associate it with the different times during fœtal life at which the various fibres of the posterior roots become medullated. The extra-medullary changes in tabes dorsalis in the peripheral nerves, the trophic changes in bones, joints and skin, are described, but the association between tabes and general paralysis of the insane is somewhat briefly dismissed. The meningitic theory of tabes, which refers the condition to compression of the posterior roots by a syphilitic affection of the membranes, is discussed, and it is pointed out that such meningitis, though frequent, is by no means constant, even in advanced cases; moreover, the symmetry of tabetic degeneration is in marked contrast to the degenerations which commonly follow meningeal affections.

The pathological appearances in muscular dystrophies, in spinal muscular atrophy, in chronic anterior poliomyelitis, in amyotrophic lateral sclerosis and in primary lateral sclerosis are then described. A distinction is drawn between progressive muscular atrophy and chronic anterior poliomyelitis, the latter being characterised by the fact that paralysis precedes muscular atrophy and is not contemporaneous with it as in progressive muscular atrophy, and also by the course of a case of chronic anterior poliomyelitis being one of definite waves rather than a gradually progressive one, and finally by the remissions which occasionally occur during its course.

After a chapter on anæmic degenerations, emboli, hæmorrhages and œdema, three lectures are allotted to the difficult subject of acute myelitis, a clinical term applied to all cord affections not confined to any particular systems or constituents of the cord, but affecting sensory, motor, trophic and reflex functions in a wholesale, irregular, non-systematic fashion. The term "myelitis" includes not only inflammatory processes, but also certain non-inflammatory ones. It is not applied to the results of traumatism, to anæmic necrosis, or to hæmorrhages or emboli. Three chief pathological types of myelitis are described, viz.:—parenchymatous degeneration, small-cell infiltration and softening. Parenchymatous myelitis is characterised by swelling and œdema of the connective-tissue elements, with degenerative changes in the nervous elements; small-cell infiltration shows a preponderance of vascular changes, of hyperæmia and of leucocytic infiltration, with occasional hæmorrhages, whilst softening consists in the reduction of a portion of the cord to a pappy or fluid consistence, the broken-down area being made up of proliferated leucocytes, together with detritus of nerve elements. This last variety is often associated with vascular thrombosis. If capillary hæmorrhages occur into such an area we have the so-called red softening. Unfortunately corresponding clinical distinctions between these different pathological forms cannot at present be drawn.

Poliomyelitis is regarded as an affection of infective origin, distributed in the area of the anterior spinal artery. Landry's paralysis is also due to an infection, such a view being supported by the concomitant enlargement which occurs in the spleen and mesenteric glands during the disease. The varying pathological appearances which have been found, tigrolysis of the anterior cornual cells, polyneuritis, &c., and the occasionally entirely negative results observed, are to be explained by the fact that the poison which produces the symptoms does not always have time to produce recognisable microscopic changes before a fatal result ensues.

After an account of acute disseminated myelitis and purulent myelitis, a description of chronic myelitis is given, and the two chief types are shown to be, one in which there is a condition of primary degeneration with consequent sclerosis, and another in which a primary softening is followed by cicatricial contraction.

The morbid anatomy of insular sclerosis is excellently described and illustrated, and the various theories regarding its pathogenesis are referred to. The writers come to the conclusion that cases of insular sclerosis are to be classified into two groups, the one a secondary degeneration following an acute disseminated myelitis of inflammatory, syphilitic or vascular origin, the other a primary form of degeneration, the result of incomplete or anomalous development, or of hypoplasia of the nerve elements.

The various combined system degenerations and the traumatic affections of the cord are discussed. Under the subject of regeneration after injuries it is pointed out that only a few new fibres are formed in the region of the lesion, and those are in close association with neurilemma cells, thus belonging to the type of peripheral rather than to that of central nerve-fibres. Spinal contusion and spinal shock are described, and chapters on compression-myelitis of tuberculous and other origin, on syphilitic and tuberculous disease of the cord, on developmental anomalies, syringomyelia and spinal tumours, conclude a volume of remarkable interest.

The book is copiously illustrated by clear and well-chosen drawings, many of them coloured, the execution of which leaves nothing to be desired. As a *résumé* of our present knowledge of the pathology of the spinal cord Drs. Schmaus and Sacki's work merits the highest praise.

PURVES STEWART.

Studii Clinici ed Anatomo-Patologici sull' Idiozia (*Clinical and Anatomo-Pathological Studies on Idiocy*). By Dr. PELLIZZI, Teacher of Psychiatry at the University of Turin. (Turin, 1901, pp. 275.)

THIS book is divided into three parts:—The first and most important part treats of idiocy with tuberose sclerosis; the second gives a classification of idiocy; and the third describes shortly epilepsy and idiocy.

Dealing with the first part, we find that idiocy with tuberose sclerosis occupies 200 out of 275 pages of which the book consists. After a short introduction, the author gives briefly an historical account of idiocy with tuberose sclerosis, in which the labours of Bourneville and others who have written on the subject are fully recognised. A description is given of twenty-two cases which the author had collected, five of them being described by Bourneville and his assistants. He then proceeds to give an account of three cases which had come under his own observation—the family history, personal examination, course of the disease, and autopsy, with the microscopical appearances seen by the Golgi and Weigert-Vassale methods, being given in each case. The second and third cases are more fully described, and the distribution of the tuberosity, the state of the neuroglia, nerve-cells, white substance, and blood-vessels of the brain and of the spinal cord are given at length. The author presents a table of the twenty-five cases of tuberose sclerosis : the name of the author comes first, then the sex and age of the patient, facts concerning hereditary predisposition and other etiological conditions, physical anomalies, the commencement of the case, the motor and psychical symptoms and the result follow, and the principal anatomo-pathological facts are summed up at the end of the table. On looking through it we find there is a history of hereditary predisposition in every case, but other etiological conditions, such as emotion, fright, and epileptic attacks of the mother during pregnancy, were only found in eleven cases. A curious fact is noticeable, viz., that the chief physical anomalies were renal tumours, which were present in sixteen of the twenty-five cases. The motor symptoms consisted entirely of epileptic attacks. With regard to etiology, the external physical and psychical causes and internal causes are mentioned, and idiocy with tuberose sclerosis is compared with other forms of the affection. The symptoms of the disease can be described in a few words. They consist of psychical and motor alterations; under the former head is arrest of mental development, which varies from semi-imbecility to the most profound idiocy; under the latter, ordinary convulsions, rarely partial epilepsy, and sometimes simply choreic movements, but no paralysis nor paresis, and no contraction of limbs. The clinical differential diagnosis between the affection under notice and sclerosis in infants, atrophic sclerosis, and epilepsy is given, and the macroscopical and microscopical appearances of the sclerosis are fully described. To the naked eye tuberose sclerosis consists of islands of sclerosis, numbering sometimes as many as thirty or forty, scattered throughout the hemisphere. They (the islands) are white or slightly grayish and shining in the peripheral parts, but towards the centre the gray is more intense in colour, and has more of a gray-yellowish tint. In consistence they are elastic at the periphery, becoming more resistant towards the centre, finally assuming a cartilaginous hardness. As regards the microscopical appearances, it should be noted that each tuberosity or hypertrophied sclerotic plate consists of nerve elements and neuroglia. In the central or hardest part, the nerve-cells are few in number and sometimes almost completely absent, but there is a dense intertwinement of fibres of the neuroglia, which are extremely slender and form a very fine network. In the peripheral parts, on the other hand, the tissue is less dense, the cells of the arachniform neuroglia are much larger and the intertwinement is looser. The nerve-cells although more scarce than normal are more numerous and less altered than in the central parts. The differential diagnosis, anatomically, between this and other forms of idiocy is fully pointed out, and an anatomical comparison with other pathological processes of the central nervous system, but unaccompanied by idiocy, such as epilepsy, multiple sclerosis, syphilitic cerebral lesions, and cerebral tumours, is made. This part of the book contains the best description of the affection, which is well known to the reviewer, that has yet been published.

In the second part the author refers to and describes the classifications of idiocy adopted by Bourneville, Ireland, Shuttleworth and the reviewer, Hammarburg and Tanzi, and proposes a classification of his own. He divides idiocy into three great classes; first, that which is due to defect of development of the brain, or idiocy of endogenous origin; secondly, that which is caused by pathological processes of the brain and its convolutions and by accident, or idiocy of exogenous origin; and thirdly, that which is due to mixed causes. These three classes with their groups and subdivisions are presented in the form of a table.

In the third part, Dr. Pellizzi quotes the opinions of the ancient and modern writers who have written on epileptic idiocy, and goes on to say that he recognises two chief forms of it, the first being idiocy with essential epilepsy, and the second idiocy with idiopathic epilepsy. He also mentions the existence of a primitive, true, endogenous idiocy with epilepsy, epileptic dementia and epileptic idiocy.

A very full bibliography is given at the end of the book, which is made more interesting by some excellent engravings, which illustrate the macroscopical and microscopical appearances which have been observed by the author in tuberose sclerosis.

The book contains much interesting information which will be useful to specialists in mental and nervous diseases, and especially to those who have to treat idiocy and imbecility.

Fletcher Beach.

An Atlas of the Medulla and Mid-brain. By Florence R. Sabin, M.D. Edited by Dr. Henry Knower. (Baltimore, 1901.)

This Atlas, described as an illustrated laboratory manual, is planned to meet the wants of the student working at practical neurology, and to afford him the means of accurately and satisfactorily studying structures by quickly comparing his own sections with the figures of the Atlas. A point of value which is observed on looking through the Atlas, and which is less commonly noticed in illustrated manuals, is the number of longitudinal and horizontal sections through the ponto-medullary region. The illustrations consist of a series of sections passing through the medulla, pons and mid-brain of a new-born infant, in the first series from the dorsal to the ventral surface, and in the second from the spinal cord upwards towards the cerebrum. Eight full-page plates at the end of the Atlas illustrate portions of Miss Sabin's well-known model of the mid and hind-brain. The Atlas will no doubt be of assistance to those requiring a laboratory manual.

W. A. Turner.

Beiträge zur Pathogenese und pathologischen Anatomie der Epilepsie. von Dr. L. W. Weber. (Jena, 1901.)

This is a short memoir upon the pathological appearances, coarse and microscopic, found in a series of thirty-five cases of true epilepsy. The results obtained by the author may be briefly stated: in many cases there is a thickening of the cortical neuroglia, partly from an increase in the number of glia corpuscles and partly from the presence of a fine network of glial fibres, the thickness and extent of which is directly proportional to the duration of the disease.

The changes in the blood-vessels of the cerebral cortex consist, in acute and recent cases, of hyperæmia, œdema of the lymph and perivascular spaces, extravasations and small-celled infiltrations; in chronic cases, of thickening of the arterial walls, and the formation of new blood-vessels in the superficial cortical layers; while in the pyramidal cells Nissl changes may be detected in old cases, pigment deposits, shrinkage of the cell bodies and falling off of the processes.

The general bearing of these observations upon the clinical types of the disease is that in congenital and early cases there is found chiefly the subpial fibrous network already mentioned, sclerosis of the blood-vessels, and disappearance of the nerve elements; while in the so-called "late epilepsy" the chief changes are irregular thickening of the cortical neuroglia with disturbance of the normal cortical structure.

There is nothing in these alterations which may not be described as consequences of epilepsy.

W. A. TURNER.

Der Farbensinn der Tiere. By W. A. NAGEL. Wiesbaden. (J. F. Bergmann, 1901, pp. 32.)

IN this lecture Professor Nagel discusses the existence and nature of the colour sense in animals. He regards protective colouration as indicating that most of the higher animals have a colour sense, but points out that zoologists are usually content to assume the existence of this sense, and do not seem to realise that the assumption is one capable of verification or disproof by exact methods.

Professor Nagel points out that one difficulty in the exact investigation of the colour sense in animals has only become apparent owing to recent advances in our knowledge of retinal physiology. In the earlier work on the subject the factor of brightness was neglected, and in reading early accounts one has no means of telling how far the reaction of an animal to a given colour was due to the colour-tone and how far to the brightness of the colour. In more recent investigations attention has been paid to this factor, but one point has been lost sight of. We know that the relative brightness of two colours may be very different to a colour-blind man and to an individual with normal colour sense. Further, the two colours may vary greatly in relative brightness for the same individual under different conditions of retinal adaptation. The possibility is suggested that the relative brightness of two colours may not be the same to different animals, and that an animal appearing to distinguish two colours, which are of the same brightness to the normal human eye, may nevertheless be guided by differences of brightness and not by differences of colour tone.

As a matter of fact, Abelsdorff has found that there is a marked difference between the pigeon and owl in the amount of constriction of the pupil produced by the action of red and blue rays, and since the amount of constriction of the pupil is, so far as we know, proportional to the brightness of a colour, we may conclude, with a considerable degree of probability, that the relative brightness of red and blue is not the same to the two birds.

Another objective measure of brightness is the action current. Nagel and Himstedt have experimented with this on the frog's eyeball, in the conditions of adaptation both to light and dark. In the former condition the largest variation was found on stimulation with yellow light from about the line "D," while the dark-adapted eyeball reacted most to green light from near the thallium line. That is to say, the maxima of action for the frog's retina in light and dark adaptation exactly corresponded with the places of maximum brightness of the spectrum as seen by the human eye in light and dark adaptation respectively. There is much reason to believe that the shifting of the place of maximum brightness is due to the different degree of sensibility of the rods and cones in the two conditions of adaptation, and it is therefore interesting to find the same shifting in the frog, whose retina also contains both rods and cones.

Nagel has also attempted to investigate the action currents in pigeons and fowls (which have few rods and no visual purple), but up to the present his efforts have been unsuccessful owing to the rapid changes which occur after enucleation.

Nagel's observations do not, of course, prove that the colour system of the frog is the same as that of man, nor do they even show that this animal has a colour sense at all, but they increase the probability of these assumptions and they show that in future experimental work on the subject one may reasonably assume that colours which are of equal brightness to the human eye will have an equivalent action, so far as brightness is concerned, on the frog's eye.

W. H. R. Rivers.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

At a Clinical Meeting held at 11, Chandos Street, on Thursday, October 31, at 8.30 p.m., the following cases were shown:—

Dr. Ferrier.—*Case of successful removal of a cerebellar tumour.*

The patient shown, Walter B., aged 12, was admitted into King's College Hospital on May 22, 1900.

He had been suffering for several months previously with headache and vomiting—symptoms which had been increasing in severity for two months previous to admission.

He had a cerebellar gait and there was well-marked double optic neuritis. The right occiput was somewhat more prominent than the left, and pressure over this region caused some tenderness. This swelling was supposed to have originated from a fall downstairs in infancy.

It was decided to trephine over this region, and the operation was performed by Mr. Burghard on June 13. The bone was found to be unusually thin, and the right lobe of the cerebellum on exposure was seen to bulge considerably, but no tumour was visible. On the lateral lobe being incised a tumour was disclosed apparently filling up the whole of the interior of the lobe. This was readily shelled out with the finger, and a tongue-like process, which extended more deeply, was cut off together with some doubtful-looking tissue in which it appeared to end. Great collapse (from hæmorrhage) followed the operation, but after transfusion of saline solution and the administration of stimulants the pulse improved and the patient made an uninterrupted recovery. The optic neuritis gradually subsided and the patient left the hospital on July 22 quite well. He has continued so since, and attends school and engages in all outdoor games like any other schoolboy.

The tumour was found on microscopical examination to have the characters of a fibro-endothelioma.

Dr. FERRIER and Dr. ALDREN TURNER.—*Case of successful removal of a cerebral tumour.*

Mrs. P., aged 34, began to have twitchings affecting the left side of the face in 1894, which continued with increasing frequency and severity into 1895, when she had two fits which affected the left limbs as well as the face. During 1898 the seizures were of frequent occurrence. In March, 1900, left hemiplegia made its appearance, and in January, 1901, optic neuritis, the fits meanwhile continuing, some of them having the character of general epileptic seizures.

She was admitted into King's College Hospital, under Dr. Ferrier's care, on July 15, 1901. On August 10 Mr. Cheatle operated and removed a small fibrous, encapsulated tumour from the lower part of the right Rolandic region.

The patient has made an uninterrupted recovery, with entire cessation of fits, disappearance of the hemiplegia and resolution of the optic neuritis.

Microscopically the tumour was found to be a fibroma.

Dr. ROBERT HUTCHISON.—*Unilateral inco-ordination, with inability to rotate the eyes downwards, in a mentally defective boy.*

Thomas M., aged 6 years 1 month, was brought to hospital for inability to walk properly. He has never walked well, but for the last six months the difficulty has been increasing, and for the same length of time it has been noticed that his head tended to be bent over towards the left shoulder. There is no history of any definite illness, but he has never been intelligent nor able to use more than one or two words; he has been well cared for but has never gone to school. There is no history of fits.

Patient is the youngest of the family. There are four brothers and two sisters alive and healthy, but two miscarriages immediately preceded the birth of this child.

Mother died, aged 42 years, after a week's illness, beginning with a fit. The father is alive and well. There is no history of syphilis. One maternal uncle died of general paralysis of the insane, and his son is at present in an asylum with the same disease.

Patient is well developed for his years, but has a vacant expression and is evidently an imbecile. He has a slight alternating squint, and the lower part of the sclerotic in the right eye is exposed from an upward inclination of the globe. He tends to keep his head a little flexed laterally to the left but without rotation, but it can be restored to the vertical position without difficulty. He presents no evidence of congenital syphilis. Muscular power in the right limbs is normal; the grasp on the left side is feeble and he often uses the right hand to raise the left arm. There is marked intention tremor with wide excursus on attempted use of the left arm. He walks with rather a wide base, but without deflection; the left leg in walking is raised high with a jerky motion, the foot thrown forward and the heel brought down sharply.

Both knee-jerks are +, especially the left; ankle-clonus not obtained; extensor response on left, flexor on right side. No tendon reflexes obtained in the upper limbs. He has complete control of his sphincters. The pupils are equal and react normally. He cannot be got to rotate the eyes downwards, and in order to see objects below the horizontal plane he bends his head forwards. The other ocular muscles are normal, but he has occasionally slight lateral nystagmus. There is no optic neuritis, and no apparent paralysis of other cranial nerves. There is no spinal curvature, and the other organs are healthy.

Dr. BEEVOR.—*A case of muscular atrophy with loss of sensation over the 1st dorsal root area.*

Ethel C., aged 19, quite well till three years ago. When 12 years old had scarlet fever; 13 years had chorea; 15 years had second attack, after which she began to have wasting of the right hand; no pain.

At present has atrophy of all the right hand muscles, with slight weakness of the flexor of fingers. Electric Reaction.—Thenar muscles do not react to faradism, interossei and hypothenar react feebly. She has a patch of anæsthesia and analgesia over area of right dorsal root.

Dr. BATTEN.—*Spasmodic movements of the limbs in an infant.*

W. M., male, aged 1½ years old, the fifth child of a family of five, of whom all the others are healthy, was born at full time in natural labour.

Was quite well till 9 months old and was able to sit up. When 9 months old had a series of fits, and since then the child has been weaker and the spasmodic movements have developed.

The child is well-nourished, is on its back and has a continuous series of irregular clonic movements of the head, arms, legs, trunk and fingers. The legs in particular are frequently suddenly flexed on the trunk, and only gradually return to their normal position. The mental condition is probably defective, there is no rigidity. Knee-jerks equal and brisk. No ankle-clonus. Plantar gives flexor response. Vision apparently good.

Dr. HEAD.—(1) *A young woman with muscular atrophy, probably of neuritic origin.*

Emily B., aged 27, single, a confectionery packer.

With the exception of several attacks of anæmia she was well until Christmas, 1899. She then noticed that her right thumb "gave in" at her work, and that the first dorsal interosseous space was wasted. She attended St. Bartholomew's Hospital during the winter of 1900, and the London Hospital since February, 1901. During the last twelve months the left hand has begun to waste.

Before the wasting in the hands began she had pains in the arms and shoulders, and she still has occasional pains which are not severe.

She now shows marked wasting of the right hand, particularly of the thenar and hypothenar group and interossei, particularly the first dorsal interosseus. In the left hand the first dorsal interosseus is wasted and the thenar muscles are smaller than normal.

On the right side the supra- and infraspinatus and the deltoid are distinctly wasted.

All the muscles of the right fore-quarter can be made to respond to faradism except the extremely wasted muscles on the outer side of the thumb; the deltoid responds feebly. To galvanism the deltoid shows a slow contraction and A.C.C. > K.C.C. In the thenar muscles there is no actual reversal, but contraction is slow. In the first dorsal interosseus A.C.C. > K.C.C.

All the muscles of the left fore-quarter react normally to faradism, but those of the thenar eminence do not respond so easily as the hypothenar muscles. To galvanism all muscles react normally, but contraction is slow in the first dorsal interosseus and the muscles of the thumb.

There is no paræsthesia or anæsthesia.

Reflexes, cranial nerve, and sphincters are normal.

Improvement has taken place under massage.

(2) *A Case for diagnosis.*

Ellis B., aged 61, a brushmaker, was admitted to the London Hospital under Mr. Roxburgh on account of pain in the left knee.

Thirty-four years before this knee had been cut with a knife, had suppurated and ankylosed in a semiflexed position. About May, 1900, the pain, which had always been present to a bearable degree, became intolerable.

All attempts to relieve this pain were ineffectual, and a Stokes-Gritti amputation was performed in October, 1900, by Mr. Barnard.

All pain ceased and he says he was never so comfortable in his life—able to do his work and to get about on crutches.

On December 22, 1900, he spent the day in the Hospital for the Christmas Entertainment. At the end of the day whilst standing he suddenly pitched forwards but did not lose his senses. His foot was found to be completely paralysed.

No history of syphilis can be obtained, and except for the ankylosed knee he has been healthy.

The left leg has been amputated and is now free from pain. Movement and muscular development of the thigh and hip good. Sensation unaffected.

All movements of the right foot are absent. He cannot move the toes or dorsiflex and plantar-extend the foot. The knee is slightly flexed, possibly from constantly sitting in a chair. Faradism evokes no response in the tibialis anticus, extensor longus digitorum, peroneus longus and soleus. With galvanism the contraction is sluggish and in the soleus A.C.C. > K.C.C.

Pain, heat and cold are not appreciated over an area that appears to be that of the first sacral.

Touch is lost over the same area, but the edges are less defined.

The knee-jerk cannot be obtained in the right knee.

Pupils react to light and accommodation.

Sphincters act normally.

Dr. MUSKENS, of the Hague, Holland, gave a lantern demonstration illustrating *Disturbances of sensation of segmental distribution in Tabetics and Epileptics.* In order to record the results as accurately as possible the patients were photographed both front and back, using different bony points as landmarks, having first examined and marked out on the skin the analgesic areas (with vertical lines) and the anæsthetic areas with horizontal lines.

Examining in this way carefully every individual case of tabes the records confirmed the view that the disturbances of sensation in locomotor ataxia really developed in segmental areas. In incipient cases he succeeded frequently in marking out, as the upper border of the thoracic area, the upper margin of the first dorsal root area quite accurately. The lumbar analgesic (or hypalgesic) area appeared not to be limited to one particular segment in incipient cases with the same regularity (photographs shown). In other incipient cases the different stages of anæsthesia had been followed out and information obtained about the way in which different forms of defective æsthesia follow upon each other. (Compare L. J. J. Muskens, "On the development of the Disturbances of Sensation, &c.," *British Medical Journal*, December 9, 1899.) In the rare cases in which there was an opportunity to examine the cord of such cases, the corresponding anatomical substratum could best be demonstrated by counting (Weigert-stain) the relative number of degenerate fibres in the posterior columns of the different spinal segments.

Subsequently, having an opportunity to examine a great number of epileptics, Dr. Muskens was struck with the fact that in a great number of these the same areas on the trunk, which were so frequently found analgesic in tabetics, presented a marked hypalgesia for pinpricks. This lack of acute sensation for pain was particularly striking in several cases which were examined a short while before a fit came on. The borders of the hypalgesic areas showed mostly a marked segmental character. A number of cases were then examined every day for a considerable period. In most cases the extent of the hypalgesic field was found to vary a great deal. In most cases where fits seldom occurred the influence of the discharge upon the extent of the hypalgesic area became very apparent. In some of these cases the discharge could be predicted with some probability for one or more days. Diagrammatic curves, demonstrating the variation in the extent and intensity of the hypalgesia, showed in many cases a marked increase in the amount of hypalgesia shortly before the fit, even up to complete and total analgesia of the body; on the other hand, some time after a fit, or a series of fits, was over, general hypalgesia was usually present over the entire skin. Generally, also, in the intervals between the discharges the curves recording the upper and lower segmental border showed a rather irregular increase and decrease of the extent of the hypalgesic area. The return of the sensibility to pain of both sides of the body appeared to be different in a number of cases; the side which discharged first and strongest, as a rule, regained its sensation last.

Dr. Muskens did not think that these hypalgesic segmental areas could be looked upon as characteristic of epileptics, although in only a few non-epileptics a similar hypalgesic area was found.

Some sensory areas showed a remarkable tenacity in regard to their sensation to pain, even in otherwise very complete pre-epileptic analgesia, and also regained their normal sensation sooner than the neighbouring parts, viz., a small field, practically coinciding with the second branch of the fifth nerve, one on the inner side of the ball of the thumb, and one occupying part of the planta pedis. These same areas appear to show a similar tenacity in regard to the sensation to pain in apoplectic and other forms of cerebral hemianalgesia.

The Meeting held at 11, Chandos Street on Thursday, December 5, at 8.30 p.m., was devoted to the consideration of Methods of Psycho-physical Research.

Dr. RIVERS explained:—(1) *Methods of estimating mental fatigue and practice.*

This subject was illustrated chiefly by an account of the results of a comparative investigation on Papuan and English children. Work for three-quarters of an hour was carried on at intervals of a day, two days, a week and a month. The influence of pauses of five minutes and a quarter of an hour during the period of work was also shown.

The full account will be published in the Reports of the Cambridge Expedition to Torres Straits.

(2) *Methods of measuring visual illusion.*

This subject was also illustrated by means of comparative work on Papuan and English observers.

Dr. MOTT and Dr. W. G. SMITH demonstrated a membrane key and a falling shutter.

The Index of BRAIN for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, is now ready.

Members of the Neurological Society can obtain the volume from Messrs. MACMILLAN & Co., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Society the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

As the cost of the Index has been considerable the Council hope that every member of the Society will order at least one copy.

EDITOR.

Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1901

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1900.

LONDON:
JOHN BALE, SONS & DANIELSSON, LTD.
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1901

Office Bearers for the Year 1901.

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CHARLES E. BEEVOR, M.D., F.R.C.P

Editor (ex-officio).

R PERCY SMITH, M.D., F.R.C.P.

Secretaries.

W. S. COLMAN, M.D. F.R.C.P
HENRY HEAD, M.D., F.R.C.P., F R.S

Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 24, 1901.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The members now number 215.

The Inaugural Address was delivered at the Annual General Meeting, on February 8th, 1900, by Dr. Augustus Waller, F.R.S., "On the Excitability of Nervous Matter, with especial reference to the Retina."

The greatest feature in the year's programme has been the delivery of the second Hughlings Jackson Lecture, on Thursday, November 29th, 1900, by Professor E. Hitzig, of Halle, who chose for the subject of the lecture, "Hughlings Jackson and the Cortical Motor Centres in the Light of Physiological Research."

It is with great regret that the Council announces that Dr. A. de Watteville has resigned the Editorship of "Brain." When accepting his resignation the following resolution was unanimously adopted: "The Council accepts with great regret Dr. de Watteville's resignation of the Editorship of 'Brain,' and desires to take this opportunity of recording the deep debt of gratitude that the Society owes him for the way in which he has conducted the Journal for the past twenty years. The Council feels that parting with Dr. de Watteville is an event of great moment to the Society, for he has not only brought 'Brain' to a high standard of perfection and secured for it a great European reputation, but even the existence of the Journal at the present time is due to his energetic action at a critical juncture in 1880. Moreover, the Council is mindful that the Society itself took origin on Dr. de Watteville's initiative, at a meeting held at his house, on November 14th, 1886."

The Council regrets the loss that the Society has sustained by the death of a distinguished member, Professor Sir Thomas Grainger Stewart, of Edinburgh, and of Mr. A. Symons Eccles.

The Council has also to report with much regret the resignation of the Senior Secretary, Dr. Risien Russell, after four years of valuable service rendered to the Society.

An abstract of the Proceedings of the Society appended to this Report shows the character and value of the work of the Society during the past year.

Proceedings of the Society during the year 1900.

February 8th, Dr. A. Waller, F.R.S., Inaugural Address, "The Excitability of Nervous Matter, with especial reference to the Retina."

March 15th, Dr. F. E. Batten, "Case of Congenital Cerebellar Defect." Dr. Farquhar Buzzard, "A Case of Cerebral Diplegia." Dr. Risien Russell, (1) "Two Cases of Thomsen's Disease (Myotonia Congenita)"; (2) "A Case of Functional Wry-neck, with Clonic Spasm of the Palate." Dr. Edwin Bramwell, "Two Cases of Myasthenia Gravis." Dr. James Collier, "A Case of Muscular Atrophy in the Upper Extremities."

May 10th, Dr. Wakelin Barratt, "Subdural Blood Cyst compressing Right Hemisphere." Dr. F. E. Batten, (1) "Series of mounted Specimens of some of the Grosser Pathological Changes found in the Nervous System"; (2) "A Cyst in the Seventh Left Dorsal Posterior Root Ganglion, from a Case in which a Subcutaneous Hæmorrhage had taken place in the Distribution of the Seventh Left Thoracic Root some months previously." Dr. James Taylor, "Sections of the Nuclei of the Third and Sixth Cranial Nerves in a Case of Ophthalmoplegia Externa." Dr. J. S. Collier, (1) "Photographs and Bone of a Case of Leontiasis Ossea"; (2) "Skull from a Case of Syringomyelia simulating Leontiasis Ossea"; (3) "Endothelioma of Skull." Dr. A. Bruce, "Tumour of Third Ventricle."

June 21st, Dr. Head, "Two Cases of Muscular Atrophy in Young Women, probably of Neuritic Origin." Dr. Colman, "A Case of 'Ophthalmoplegic Migraine.'" Dr. Lees, (1) "Hæmatomyelia"; (2) "Hysterical Contracture." Dr. Leonard Guthrie, "Thomsen's Disease." Dr. Newton Pitt, (1) "Insular Sclerosis"; (2) "Two Cases of Doubtful Nature, possibly Insular Sclerosis."

October 25th, in the Physiological Laboratory, St. Mary's Hospital, Mr. Victor Horsley, F.R.S., (1) "Note on the Course of the Fillet Fibres in the Cat"; (2) "Further Note on a Cat in which Brown-Sequard Fits were obtained after a Lesion of the Medulla Oblongata, the Fits being still obtained after Complete Division of the Mesencephalon." The President, "The Excitability of Nervous Matter—its Last Sign of Life."

November 29th, the Second Hughlings Jackson Lecture, Professor Eduard Hitzig, of Halle, "Hughlings Jackson and the Cortical Motor Centres in the Light of Physiological Research."

BALANCE SHEET, 1900.

Cr. NEUROLOGICAL SOCIETY. **Dr.**

RECEIPTS.

	£	s.	d
To Balance	45	9	2
Subscriptions	230	3	0
Half Share of profit on "Brain" ...	34	19	4
Contribution by Messrs. Macmillan towards cost of Illustrations in "Brain"	11	13	0
	£322	4	6

EXPENDITURE.

				£	s.	d.
By Stationery and Printing—Bale & Danielsson				27	4	8
Illustrations—Bale & Danielsson ..				76	0	0
Microscopes and Lanterns—						
C. Baker	£7	2	0			
A. Hawes	2	4	6			
				9	6	6
Hire of Rooms and Refreshments—Medical Society				20	15	0
Messrs. Macmillan for publishing "Brain"				121	0	0
Bank Charges				0	4	0
Petty Cash				1	13	0
Balance ...				66	1	4
				£322	4	6

Audited and found correct December 5, 1900,

H. G. TURNEY,
FREDERICK E. BATTEN.

CHARLES E. BEEVOR, *Hon. Treas.*

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorar Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of ' BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by R. Percy Smith, M.D. F.R.C.P.

HONORARY MEMBERS.

Mr. Herbert Spencer, London.
Professor Wundt, Leipzig.
,, Hitzig, Halle.
Sir Samuel Wilks, Bart., London.

CORRESPONDING MEMBERS.

Professor Erb, Heidelberg.
,, Golgi, Pavia.
,, Goltz, Strasburg.
,, Grasset, Montpellier.
,, Jolly, Berlin.
,, Luciani, Rome.
,, Munk, Berlin.
,, Obersteiner, Vienna.
,, Retzius, Gustaf, Stockholm.
,, Ribot, Paris.
Dr. Weir Mitchell, Philadelphia.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886	J. Hughlings Jackson, M.D., F.R.S.
1887	Sir Samuel Wilks, Bart., M.D., F.R.S.
1888	Sir James Crichton-Browne, M.D., F.R.S.
1889	Jonathan Hutchinson, F.R.S.
1890	Thomas Buzzard, M.D.
1891	John S. Bristowe, M.D., F.R.S.
1892	H. Charlton Bastian, M.D., F.R.S.
1893	Edward A. Schafer, F.R.S.
1894	David Ferrier, M.D., F.R.S.
1895	Sir William H. Broadbent, Bart., M.D., F.R.
1896	Alexander Hill, M.A., M.D.
1897	George H. Savage, M.D.
1898	Victor Horsley, M.B., F.R.C.S., F.R.S.
1899	W. H. Gaskell, M.D., F.R.S.
1900	Augustus Waller, M.D., F.R.S.
1901	W. Julius Mickle, M.D.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897	J. Hughlings Jackson, M.D., F.R.S.
1900	Professor Eduard Hitzig, Halle.

⁂ Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 Abrahams, Bertram, M.B., M.R.C.P., 14, Welbeck Street, W.

1892 Alexander, Robert Reid, M.D., The Asylum, Hanwell, W.

1897 Anderson, H. Kerr, M.B., B.Sc., 9, Park Terrace, Cambridge

1894 Andriezen, W. Lloyd, M.D., 7, Apsley Terrace, Acton, W.

1891 Ballance, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)

O.M. Barlow, Sir Thomas, Bart., M.D., F.R.C.P., 10, Wimpole Street, W.

1897 Barratt, J. O. Wakelin, M.D., F.R.C.S., Pathological Laboratory, West Riding Asylum, Wakefield.

O.M. Bastian, H. Charlton, M.D., F.R.C.P., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.-P., 1891; C., 1886-89, 1893-95.)

1897 Batten, Frederick E., M.D., M.R.C.P., 124, Harley Street, W.

O.M. Beach, Fletcher, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, Beevor, C. E., M.D., F.R.C.P., 33, Harley Street, W. (Tr., 1894-1901; C., 1893.)

1888 Birt, Ernest, M.D., West Riding Asylum, Wakefield.

1898 Bolton, Joseph Shaw, M.D., B.S., B.Sc , Claybury Hall, Woodford, Essex.

1896 Bond, C. Hubert, M.D., D.Sc., London County Asylum, Bexley, Kent.

1899 Bond, J. W., M.D., B.S., 26, Harley Street, W.

1892 Bowlby, Anthony, A., F.R.C.S., 24, Manchester Square, W.

1898 Boyce, Rubert W., M.B., University College, Liverpool.

,, Boycott, Arthur Norman, M.D., Hertfordshire County Asylum, Hill End, St. Albans.

,, Bradbury, John B., M.D., F.R.C.P., 4, St. Peter's Terrace, Cambridge.

1889 Bradford, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

O.M. Bramwell, Byrom, M.D., F.R.C.P.Ed., 23 Drumsheugh Gardens, Edinburgh. (C., 1894

Elected

1899 Bramwell, Edwin, M.B., C.M., M.R.C.P., 2, Randolph Place, Edinburgh.

1895 Bramwell, John Milne, M.B., C.M., 2, Henrietta Street, W.

1892 Bristowe, Hubert Carpenter, M.D., Wrington, Somerset.

O.M. Broadbent, Sir Wm. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.-P., 1893, C., 1886-87, 1896.)

1894 Brodie, T. Gregor, M.D., 55, Marlborough Mansions, West Hampstead, N.W.

1897 Brown, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 Brown, Sanger, M.D., 100, State Street, Chicago.

O.M. Bruce, Alex., M.D., F.R.C.P.Ed., 8, Ainslie Place, Edinburgh. (C., 1895.)

,, Bruce, J. Mitchell, M.D., F.R.C.P., 23, Harley Street, W.

1898 Bruce, Samuel Noble, M.R.C.S., 15, Queensboro' Terrace, Hyde Park, W.

O.M. Brunton, Sir T. Lauder, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)

1895 Bryan, Frederick, M.B., Horton Manor Asylum, Banstead Downs, Surrey.

,, Bryant, John Henry, M.D., M.R.C.P., 8, Mansfield Street, Portland Place, W.

1890 Bury, Judson Sykes, M.D., F.R.C.P., 10, St. John Street, Manchester.

1900 Buzzard, E. Farquhar, M.B., M.R.C.P., 137, Bedford Court Mansions, W.C.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-89; C., 1886-87, 1891-95.)

1898 Caley, H. A., M.D., F.R.C.P., 24, Upper Berkeley Street, W.

,, Campbell, Harry, M.D., F.R.C.P., 23, Wimpole Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1899 Chambers, James, M.A., M.D., The Priory, Roehampton.

1891 Clarke, J Michell, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

1898 Clinch, Thomas Aldous, M.D , 16, Regent Street, Nottingham.

1892 Cole, Robert Henry, M.D., M.R.C.P., 48. Upper Berkeley Street, Portman Square.

1899 Collier, J. S., M.D., B.Sc., M.R.C.P., 57a, Wimpole Street, W.

1890 Colman, W. S., M.D., F.R.C.P., 22, Wimpole Street, W. (Sec., 1899-1901.)

,, Corner, Harry, M.D., Brook House, Southgate, Middlesex.

1899 Coupland, Sidney, M.D., F.R.C.P., 16, Queen Anne Street, W.

1895 Davidson, Andrew, M.D., The County Asylum, Dorchester, Dorset.

1897 Dawson, William, R., M.D., F.R.C.P.I., Farnham House, Finglas, Co. Dublin, Ireland.

Elected

1894 Dean, H. Percy, M.S., F.R.C.S., 69, Harley Street, W.

1898 Dodd, Henry Work, F.R.C.S., 136, Harley Street, W.

O.M. Dreschfeld, Julius, M.D., F.R.C.P., 3, St. Peter's Square, Manchester. (V.P., 1898-99.)

,, Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1899 Fawcett, John, M.D., M.R.C.P., 24, St. Thomas Street, S.E.

O.M. Ferrier, David, M D., F.R.C.P., F.R.S., 34, Cavendish Square, W., (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)

,, Fox, E. Long, M.D., F.R.C.P , Church House, Clifton, Bristol.

,, Gaskell, W. H , M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.P., 1897-98; C., 1896, 1900.)

,, Gee, Samuel J., M.D., F.R.C.P , 31, Upper Brook Street, W.

,, Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1899 Goodall, Edwin, M.D., M.R.C.P., Joint Counties Asylum, Carmarthen.

1898 Goodliffe, John Henry, M.D.Aberd., Norton Hall, near Sheffield.

1897 Gossage, Alfred Milne, M.B., M.R.C.P., 54, Upper Berkeley Street, W.

1892 Gotch, Francis, M.A., F.R.S , The Lawn, Banbury Road, Oxford.

1893 Grant, J. Dundas, M.D., F.R.C.S., 18, Cavendish Square, W.

O.M. Gunn, R. Marcus, M B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 Guthrie, Leonard, M.D., F.R.C.P., 15, Upper Berkeley Street, W.

1898 Halliburton, William Dobinson, M.D., F.R.C.P , F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 Hanbury, William R., M.R.C.S., L.R.C.P., County Asylum, Brentwood, Essex.

1897 Harris, Wilfred J., M.D., M.R.C.P., 19, Oxford Terrace, W.

1898 Hartley, W. Darley, M D., M.R.C.P., Park House, Kloof Street, Cape Town, South Africa.

,, Haslett, John Hanfield, M.R.C.S., L.R.C.P , Halliford House, Sunbury-on-Thames.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.D., D.Sc., F.R.S.E., 1, St. Andrew's Place, Cardiff.

1891 Head, Henry, M.D., F.R.C.P., F.R.S., 61, Wimpole Street, W. (C., 1900, Sec. 1901.)

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896, V.P., 1895; C., 1892-94.)

Elected

O.M. HORSLEY, VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. Pres., 1898; V.P., 1896-97; C., 1892-93, 1899.)

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.B., C.M., South View, Montagne Road, Felixtowe.

1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., Lensfield, Cambridge.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W (Pres., 1889; V.P., 1887-88, C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3 Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887, 1892.)

,, JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 61, Wimpole Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W.

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., M.R.C.P., 81, Harley Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge. (V.P., 1901; C., 1898-99.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1900, 1901.)

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., The Manor Asylum, Horton, Epsom.

1899 LORD, JOHN R., M.B., C.M., London County Asylum, Bexley, Kent.

1897 LOEWENTHAL, MAX, M.D., M.R.C.P., 126, Princes' Road, Liverpool

1898 MACCORMAC, JOHN M'GEE, M.D., 31, Victoria Place, Belfast.

,, MCALDOWIE, ALEXANDER M., M.D., 6, Brook Street, Stoke-on-Trent.

1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, MACKENZIE, HECTOR WILLIAM GAVIN, M.D., F.R.C.P., 34, Upper Brook Street, Grosvenor Square, W.

O.M. MACKENZIE, STEPHEN, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896-98.)

1896 MACKINTOSH, ASHLEY W., M.A., M.D., 9, Bon Accord Square, Aberdeen.

1899 MACLULICH, PEERS, M.D., B.Ch., Joint Counties Asylum, Carmarthen.

1889 MACPHAIL, S. R., M.D., Borough Asylum, Rowditch, Derby.

Elected	
O.M.	Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)
,,	Mann, J. Dixon, M.D., F.R.C.P., 16, St. John's Street, Manchester.
1894	Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.
1891	May, W. Page, M.D., M.R.C.P., Helouan, Cairo, Egypt.
1890	Menzies, William Francis, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.
O.M.	Mercier, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1901.)
1897	Meyer, Adolf, M.D., Clark University, Worcester, Mass., U.S.A.
O.M.	Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V.P., 1900; C., 1895-97.)
1895	Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.
O.M.	Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.
1888	Mott, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1901.)
1899	Mould, Gilbert E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.
1894	Murray, George R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.
1889	Murray, H. Montague, M.D., F.R.C.P., 25, Manchester Square, W.
O.M.	Nettleship, Edward, F.R.C.S., 5, Wimpole Street, W.
,,	Nicolson, David, C.B., M.D., M.R.C.P.Ed., Balgownie, Guildford, Surrey.
1888	Niermeyer, J. H. H., M.D., Amsterdam.
,,	Norman, Conolly, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.
1887	Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.
,,	Ogilvie, Leslie, M.B., M.R.C.P., 46, Welbeck Street, W.
,,	Oliver, Thomas, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.
O.M.	Orange, Wm., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.
,,	Ord, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)
,,	Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)
,,	Orr, David, M.B., County Asylum, Prestwich, Manchester.
,,	Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (V.P., 1900-1; C., 1891-92.)
1895	Parkinson, John Porter, M.D., M.R.C.P., 57, Wimpole Street, W.
1901	Parsons, John Herbert, M.B., B.Sc., 28, Great Ormond Street, W.C.
1901	Passmore, E. S., M.D., M.R.C.P., The Asylum, Banstead.
1896	Paterson, Donald Rose, M.D., M.R.C.P., 18, Windsor Place, Cardiff.

Elected

1895 Pinder, George, M.D., B.C., Hazeldene, Farncombe Road, Worthing.

1899 Piper, Francis P., M.B., M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

1888 Pitt, G. N., M.D., F.R.C.P., 15, Portland Place, W.

O.M. Poore, G. Vivian, M.D., F.R.C.P., 24a, Portland Place, W. (C. 1898-99.)

,, Purves, W. Laidlaw, M.D., 20, Stratford Place, W.

1899 Rambaut, Daniel F., M.D., B.Ch., Richmond District Asylum, Dublin.

1894 Ransom, William B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 Rayner, Henry, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W

O.M. Reid, E. W., M.B., F.R.S., University College, Dundee.

1897 Reissmann, Charles H., M.B., B.Sc, 7, Upper Montague Street, Russell Square, W.C.

1893 Rennie, George E., M.D., M.R.C.P., College Street, Sydney.

1895 Reynolds, Ernest S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1892 Richards, Joseph Peeke, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 Rivers, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1898 Robertson, William Ford, M.D., 7, Hill Square, Edinburgh.

1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

,, Ruffer, Marc Armand, M.D., Alexandria, Egypt.

1889 Russell, J. S. Risien, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-1900; C., 1901.)

1899 Russell, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

O.M. Sanderson, Sir J. Burdon, Bart., M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.P., 1895; C., 1886-90, 1898.)

1888 Savill, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. Schäfer, E. A., F.R.S., University of Edinburgh (Pres., 1893; V.P., 1892, C., 1886-98.)

,, Semon, Sir Felix, M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)

1898 Seward, Wm. Joseph, M.B., Colney Hatch Asylum, N.

O.M. Sharkey, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 Shuttleworth, George, E., M.D., Ancaster House, Richmond.

1898 Simpson, Francis O., L.R.C.P., M.R.C.S., Rainhill Asylum, Liverpool.

1900 Singer, H. D., M.D., National Hospital, Queen Square, W.C.

1895 Smith, F. J., M.D., F.R.C.P., 138, Harley Street, W.

Elected	
1887	Smith, R. Percy, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901.)
1895	Spicer, W. T. Holmes, M.D., F.R.C.S., 47, Welbeck Street, W.
,,	Stansfield, Thos. E. K., M.B., London County Asylum, Bexley, Kent.
1895	Stanley, Douglas, M.D., 19, Easy Row, Birmingham.
1892	Starling, Ernest Henry, M.D., F.R.C.P., F.R.S., 8, Park Square, N.W.
1897	Stewart, Purves, M.A., M.D., M.R.C.P., 133, Harley Street, W.
,,	Stirling, Wm., M.D., D.Sc., Owens College, Manchester.
1894	Stodart-Walker, A., M.B., F.R.C.P.Ed., 30, Walker St., Edinburgh.
1897	Stoddart, Wm. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.
1895	Stout, G. F., M.A., St. John's College, Cambridge.
1887	Suckling, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.
1889	Sutherland, Henry, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.
1888	Syers, Henry W., M.D., M.R.C.P., 75, Wimpole Street, W.
1898	Taylor, F. R. P., M.D., Darenth Asylums and Schools, Dartford, Kent.
1891	Taylor, James, M.D., F.R.C.P., 49, Welbeck Street, W. (C. 1900-1.)
1895	Telford-Smith, T., M.A., M.D., South Hill Park, Bromley, Kent.
1898	Thomson, H. Campbell, M.D., M.R.C.P., 34, Queen Anne Street, W.
1889	Thorburn, Wm., F.R.C.S., The Limes, Nelson Street, Manchester. (C. 1901.)
1892	Titchener, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.
O.M.	Tooth, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)
1892	Trevelyan, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.
1892	Tuckey, Charles Lloyd, M.D., 88, Park Street, Grosvenor Square, W.
1892	Tuke, Sir J. Batty, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.
1891	Tuke, Thomas Seymour, M.B., Chiswick House, Chiswick.
,,	Turner, William Aldren, M.D., F.R.C.P., 13, Queen Anne Street, W.
1898	Turney, Horace G., M.D., F.R.C.P., 68, Portland Place, W.
O.M.	Tweedy, John, F.R.C.S., 100, Harley Street, W.
1888	Voorthus, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.
O.M.	Waller, Augustus, M.D., F.R.S., 16, Grove End Road, N.W. (Pres., 1900, V.P., 1899; C. 1894-97, 1901.)
,,	Ward, J., D.Sc., Trinity College, Cambridge.
,,	Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.
1892	Washbourn, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

Elected

O M. Watteville, A. de, M.A., M.B., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1900; Sec., 1886-89; Editor of *Brain*, 1880-1900.)

1897 Weber, F. Parkes, M.D., F.R.C.P., 19, Harley Street, W.

" West, Samuel, M.D., F.R.C.P., 15, Wimpole Street, W.

1897 White, Ernest W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. White, W. Hale, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1901.)

1894 Whiting, Arthur J., M.D., M.R.C.P., 142, Harley Street, W.

1889 Wiglesworth, Joseph, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1900 Williams, Leonard, M.D., 8, York Street, Portman Square, W.

1894 Williamson, Richard T., M.D., F.R.C.P., 294, Oxford Road, Manchester.

1896 Wilson, Albert, M.D., M.R.C.P., Minto House, South Woodford, Essex.

" Wood, Guy M., M.B., M.R.C.P., 6, Woburn Square, W.C.

1889 Wood, T Outterson, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed., 6, Scrope Terrace, Cambridge.

1899 Wright, Hamilton, M.D., Pathological Institute, Selangor Straits Settlement.

1898 Young, Robert Arthur, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.